THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

APRIL, 1898.

ALBUMINURIA IN LIFE INSURANCE.[1]

By Brandreth Symonds, A.M., M.D.,
MEDICAL EXAMINER OF THE MUTUAL LIFE INSURANCE COMPANY OF NEW YORK.

By the term albuminuria is meant a solution of serum-albumin and serum-globulin in the urine. No other proteids should be included. The proportion of these two substances varies in different cases, but when one is present the other is also. Therefore, hereafter, when we speak of albumin in the urine, let us remember that for the purposes of this paper we include both serum-albumin and serum-globulin. The sources of the albumin may be quite various. When the urine is mixed with blood, it always contains albumin. This blood may come from menstruation or from any part of the genito-urinary tract. Similarly, the presence of pus, unless in very small quantity, will cause an appreciable amount of albumin in the urine. Albuminuria may, therefore, originate in the urethra, the bladder, the ureters, the kidneys, or even in the genital appendages. By far the most common source is the kidneys, and in this paper the term albuminuria will be limited strictly to that of renal origin. The quantity of albumin varies from the faintest trace, shown only by the most delicate tests, to an amount equal to 1 or 2 per cent. of dry albumin. The quantity is not at all indicative of the gravity of a case. Many mild and curable cases show much albumin, while, on the other hand, it is well known that one form of chronic Bright's disease seldom has more than a trace, and occasionally none at all.

Following the old story, let us now "catch our rabbit." The tests for albumin are many, and their number increases daily. When the amount

[1] Read before the Section on Practice, New York Academy of Medicine, January 18, 1898.

is more than a decided trace, sufficient in fact to give an immediate reaction in Heller's test, the problem is simple. But when the amount is less than this it becomes more complicated and fraught with many chances for mistakes. Some of the tests are very delicate, indicating one part of albumin in two or even three hundred thousand. This leaves nothing to be desired on that score, but unfortunately the agents employed in such tests are apt to react with other substances which may be present in the urine. The most common cause of error is undoubtedly nucleo-proteid. This is a proteid probably arising from cell-disintegration, and in my experience it is very frequently found in the urine, usually in small quantity. Up to a few years ago it was confounded with mucin, owing to its viscosity and some other physical properties. But Halliberton, in the *Goulstonian Lectures for* 1893,[1] makes the following statement:

"More recently still it has been shown that the mucinoid substance in the mucus of the urine is not mucin at all, but another nucleo-albumin shed out from the cells of the urinary tract. The resemblance of mucin to the nucleins and nucleo-albumins has been a stumbling-block from the very commencement, and the solubilities of the two substances are so similar that there will always be a liability to confuse them."

It is readily precipitated by nearly all organic acids, either diffused or by a contact-test. It is also precipitated to some extent from acid urine by boiling. These statements cast grave doubts upon the accuracy of most tests for albumin. I have tried nearly all, and do not hesitate to say that those which show marked delicacy of reaction have obtained this at the expense of accuracy. In fact, I do not know of a single test which is an infallible indicator of albumin. Sweeping as this statement is, I am prepared to defend it after a long and at times painful experience. Let us pray for the speedy coming of that bright genius who will devise a test for albumin that is at once simple, delicate, and accurate. My own practice is to use a combination of two tests. Unless the urine is distinctly acid, it should first be made so by adding two or three drops of acetic acid. The upper layer in a large test-tube is then boiled and compared in good daylight against a dark background with the lower layers. A very slight opacity in the upper layers can readily be detected. As this is my delicate test, if no reaction is given I lay the specimen aside, satisfied that there is present no albumin of clinical importance. If a reaction does show, a few drops of nitric acid are added. If the opacity does not then disappear entirely, we can be certain of the presence of albumin or nucleo-proteid. In order to differentiate these, I resort to the well-known Heller's test. In spite of the gibes

[1] British Medical Journal, 1893, i. p. 575. In a later communication in the Journal of Physiology, 1895, xviii. p. 306, he calls this class of bodies nucleo-proteids, and that expression seems to be preferable.

and flings made at this by most analysts, it is probably used more than all the other tests. I think this popularity can be attributed to its accuracy, as the sharply defined contact-reaction with albumin is only simulated with certain oleoresins. As ordinarily applied, it is not very delicate. It becomes much more valuable if we let it stand for twenty minutes or half an hour, and is then but little inferior to the most delicate tests. Instead of the boiling, I sometimes use the potassium ferrocyanide test, or I may use both. But I always check the results, if positive, with Heller's test, and depend largely, though not entirely, upon this for an affirmative reply in a questionable case of albuminuria. I have no doubt that five years ago I called most nucleo-proteid true albumin, and condemned many a man to exile or death. During these last eight or nine years I have had a very large practice in testing urine, and theoretically my ability to determine the infinitesimal trace of albumin should have increased. I do not think my hand has lost its cunning or my eye its sharpness. Nevertheless, the proportion of cases of albuminuria has fallen from 10 per cent. eight years ago to 5.5 per cent. in the first six months of 1897. It is true that eight years ago we were in the midst of an epidemic of influenza, and this must be regarded as a frequent cause of temporary albuminuria. But, even making allowance for this, the lessened frequency of albuminuria in my experience should be considered as due to my increased ability to differentiate true and false reactions.

Clinically, cases of albuminuria can be divided into two great classes, temporary and permanent. The temporary, or transient, cases can be further divided into organic and functional. Of course, the use of the word "functional" in this connection can be disputed, as has often been done, and undoubtedly there is no sharp line dividing the two sets of cases. But the classification is a convenient one, and understood by most without the need of elaborate definition. Among the organic cases of transient albuminuria, the varieties of acute nephritis furnish the most marked examples. These are occasionally primary, but usually secondary to some other disease. The recovery from them is frequently prompt and complete, leaving behind no more sequels than are left after acute lobar pneumonia. But these are not the cases one often sees applying for life insurance. Most of the latter would be classified as functional, and can be subdivided into several varieties:

1. *The cyclical variety.* In this the albumin is very scanty, or absent entirely, in the urine first passed in the morning. It then increases, slowly or rapidly, till a certain time, usually in the forenoon, and thereafter becomes somewhat less during the rest of the day, and sometimes disappears entirely. These cases are frequently anæmic and dyspeptic, generally nervous and out of sorts. It is a curious fact that most cases of Bright's disease show a daily variation in the amount of albumin,

and the urine passed in the morning immediately after rising usually contains much less albumin than that secreted at other times. For this reason I never use such urine for analytical purposes in those cases.

2. *The dietetic variety.* In this the albuminuria is supposed to be caused by certain articles of food, especially the highly nitrogenous, such as milk, eggs, etc. I have never seen a case which I felt was certainly in this class. In some cases recurrent dyspepsia seems to have been associated with recurrent albuminuria, but the latter was not dependent on the particular food eaten.

3. *The muscular variety.* The albuminuria in this is caused by severe muscular exercise. The quantity of albumin varies, and is sometimes considerable. It comes on very soon after the exercise, and lasts usually for days, sometimes weeks. In this era of football and bicycling, it is quite common. I have usually found it in persons under thirty years, probably on account of its origin ; but it may occur in those much older.

4. The albuminuria of adolescence, as its name implies, is that variety found in the young between the ages of fifteen and twenty-two or twenty-three years. It is quite common among boys, to whom my experience has been limited. These boys are sometimes anæmic and below par, but frequently they are in robust health apparently. Sometimes the urine is concentrated and loaded with urates, as Da Costa has shown ; but in many cases it seems to be normal except for the presence of albumin. The albuminuria may persist in these cases for a long time without causing permanent trouble. In one case I first noticed albumin at the age of seventeen. The boy was tall and slender, and, though not robust, was hardy and did all that boys of that age usually do in the line of sports, exercise, exposure, and various small indiscretions. After persisting steadily for two years, the albuminuria gradually improved and then assumed the muscular type. It lasted in that form for about three years, and then disappeared entirely three years ago. Although I have repeatedly examined the urine since then, I have not found any albumin. As this is frequently the age of aggressive sexual development, there is apt to be present congestion of the prostate and other accessory genital organs, with the consequent elaboration of much nucleo-proteid. Hence we must always be on our guard against a mistake in analysis in this particular class of cases.

5. A variety of albuminuria, apparently due to the irritation of concentrated urine, may occur at older ages. Kidneys in perfect health, however, seem to stand great concentration of urine without leaking albumin. Even though the analogy does not hold, it is well to remember that birds and reptiles secrete a urine so concentrated that it is practically solid. On the other hand, the nature of the irritant doubtless is a factor. Thus the persistent excretion of saccharine urine frequently causes albuminuria, and sometimes even chronic nephritis.

6. Another variety of transient albuminuria is that which is associated with influenza and ordinary colds. This is quite common in my experience. It usually lasts only a short time, disappearing a few days after the acute febrile disturbance has ceased.

I have no doubt that there are other varieties of transient albuminuria beside these which have been mentioned. There certainly are many cases which cannot be arranged under any of these classes, and some of them seem to have no cause whatever.

What is the clinical significance of the albuminuria in these numerous varieties which we have grouped together as transient? In the first place, I think we should regard all albuminuria, whether transient or permanent, as pathological. Some authorities speak of physiological albuminuria, but that is a misnomer. Albuminuria is no more physiological than a heart-murmur of anæmia, or an intermittent pulse, or an acute bronchitis. Any of these conditions can disappear promptly, and thoroughly, leaving the individual in sound health; but while they exist, no one would call him in a normal physiological condition. So with albuminuria. It may disappear entirely and permanently, and never recur, but when present the condition is abnormal. If we fail to find in many of these cases any evidence of inflammation or other organic change of the kidney, we must still regard the condition as one of disease, although our knowledge of them should prevent us from making too dismal a prognosis, as was done in the days when albuminuria was synonymous with Bright's disease.

Have we any means of distinguishing the mild cases with a good prognosis, from the more serious forms? I know of none except the continued absence of albumin from the urine. In other words, as long as albuminuria is present, I fear the possibilities of the case developing some serious organic mischief. Do not let us forget, either, the wonders that treatment can accomplish in Bright's disease for a limited time. We have all seen cases in which albumin disappeared from the urine for months under careful diet and regimen and suitable medication. On the other hand, there are certain conditions, coexistent with apparent good health, which must be regarded as potential factors for the bad in cases which we may be tempted to consider functional.

1. The longer the albuminuria persists, the greater is the probability of its organic origin. If it has lasted continuously for more than a year, the prospect is very doubtful, but not hopeless, even then.

2. A continuous albuminuria is of more serious import than one which comes and goes, especially if we can in the latter case assign some cause which would put it among the classes previously enumerated.

3. The presence of tube-casts is usually looked upon as indicative of an organic renal trouble. Still, some authorities have reported casts in cases which they regarded as of functional origin, and these cases have

usually belonged to the variety called muscular. Besides this, Kossel has endeavored to prove that casts are perfectly innocuous at times. In spite of these attempts, in life insurance we welcome them as cheerfully as we would a rattlesnake. The presence of red blood-cells, if it can be demonstrated that they are from the kidneys, is quite conclusive of organic changes there. Some observers claim that they can differentiate renal epithelial cells with certainty from those of the bladder and ureters. Those who can perform this feat are entitled to our great respect. How they recognize them I have never been able to make out, although I have diligently analyzed the rather vague differential points laid down.

4. The persistence of a specific gravity lower than a thousand and fifteen is a bad prognostic in any case of albuminuria. It is apt to indicate a renal insufficiency, and these cases are much less amenable to treatment than those in which we find the specific gravity a thousand and twenty-five or higher. If we further demonstrate renal insufficiency by determining that the amount of urea daily excreted is less than normal, the indication, of course, is still more serious.

5. A hard pulse with an accentuated sound of closure of the aortic valves is distinctly prejudicial. An increased arterial tension is thus shown which may indicate the beginning of arterio-capillary fibrosis, or which may be due to the stimulation and consequent narrowing of the arterioles by blood from which the irritant excrementitious products have not been properly removed. In either case, the sign is a bad one, as it points to something more than a functional disturbance.

6. Headaches do not occur in perfect health, and, therefore, mention of them may be considered outside the limit of this paper. They are so common, however, that I wish to draw attention to them when associated with albuminuria. They must be regarded in this connection as an evil omen, if they are at all frequent or severe.

7. The age of the patient is a point of much consequence. The younger the case, down to childhood, the more apt it is to be a functional case, and the better the chances of speedy and complete recovery. If the age is over forty, the prospects are much dimmed, and I regard such cases with suspicion, which increases with every year, both of age and my experience. The highest age at which I have found albuminuria in a man otherwise apparently healthy has been seventy-two. He was sound in all respects but this, and had no ear-marks of organic disease, but two tests on successive days showed the presence of a trace of albumin. Unfortunately, I lost track of him, and cannot say what the outcome was.

8. Extremes of weight, both high and low, are prejudicial in albuminuria, as in most other diseased conditions. We are apt to find light weight associated with albuminuria of the young, and in many of these

cases it seems to be due to the latter. On the other hand, over-weight with albuminuria is apt to occur in persons over thirty, and is, I think, of graver significance than under-weight, as the same causes which have produced the obesity have probably occasioned also organic changes in the kidney.

9. The use of alcoholics beyond the most moderate extent is unfavorable in cases of albuminuria. Certainly, if the amount taken is in the neighborhood of Anstie's limit, or if there are occasional excesses, the prospects are not good for the ultimate disappearance of the albumin.

These are the factors which have occurred to me as influencing the prognosis in cases of albuminuria. With the possible exception of headaches, they are all consistent with good health, and they might all be present in an individual who was still apparently in good health. Of course, the complication of albuminuria with any disease is serious, but with that our discussion has nothing to do.

There remains still one class of cases which come within our definition, and which must now be considered. I refer to those cases in which the albuminuria is steadily persistent, although the individual is apparently in good health otherwise. We have all met such cases, and they are certainly very puzzling. On account of these the cry of "physiological" albuminuria has been loudest raised, and here it is most difficult to meet. In these the amount of albumin is usually reported as very small, or a trace, and most of them are doubtless cases in which nucleo-proteid has been mistaken for genuine albumin. But this is not so of all, and an ingenious hypothesis (I have tried in vain to recall where or by whom first advanced) endeavors to account for them. It is as follows: An acute nephritis has occurred and passed away, leaving a limited portion of one or both kidneys altered. This lesion is permanent, but non-progressive. The sound portion of the kidneys is capable of doing all the excretory work necessary, and does it; while the damaged portion permits the constant transudation of albumin. This is certainly very plausible, but is unsupported by any investigations post mortem, so far as I know. I do not offer this theory as an excuse for the statement that I regard all these cases as pathological. They are not healthy, however much they may seem to be. They may not have any organic lesion of the kidney, and they may not die of any functional disease of that organ; but in our work we must look further than this. Do they as a class live as long as they would if entirely healthy? If some can recall cases of albuminuria which have lasted for ten or fifteen years, we must remind them that the expectation of life at thirty is thirty-five years, at forty it is twenty-seven years, and even at fifty it is twenty years. If pneumonia, erysipelas, typhoid fever, or any serious disease takes hold of such a case, is his chance of recovery as good as if he had been sound? I, for one, will give a negative reply to both these ques-

tions. But these cases of permanent albuminuria must not be confounded with those in which the albuminuria is transient. Here the prognosis is good, and as soon as the albuminuria disappears and stays away for a reasonable length of time we can regard such a case as healthy as if he had had simply an attack of functional dyspepsia.

It has been suggested that all cases of albuminuria could be insured by charging them all an extra premium, which could be so equalized that no loss would accrue to the company. This is undoubtedly true; but think how manifestly unjust it would be to the cases of transient albuminuria, who would thus be forced to pay extra, when in a few months or even weeks they might be considered in such good health as to be charged only the regular rates. It would be like accepting all cases of cough at an extra premium—a splendid opportunity for consumptives and cases of emphysema, but very harsh treatment for ordinary colds and acute bronchitis.

AMŒBIC DYSENTERY.[1]

By H. F. Harris, M.D.,
ASSOCIATE PROFESSOR OF PATHOLOGY, JEFFERSON MEDICAL COLLEGE.

Since the announcement of the discovery of the amœbæ coli in the United States by Osler, quickly followed by confirmatory reports from various sources of the presence of these organisms in the fecal discharges in cases of chronic dysentery, the subject of amœbic dysentery has assumed considerable importance. Lambl[2] was the first observer who noted the occurrence of rhizopods as human parasites, but his descriptions of the organisms which he observed show beyond doubt that they differ in many essential particulars from the one subsequently described by Loesch[3] and others. He found amœboid bodies and other members of the above group in the mucus from the bowel of a child dead of enteritis in 1859. Following this studies were made by Loesch, who found amœbæ in the stools in a case of ulcerative inflammation of the colon. This case, which was in every way a typical one, originated in St. Petersburg. He produced the disease in a dog by rectal injections of the fresh stools from this case. Kartulis,[4] in Egypt, found amœbæ present in the stools of over 500 cases of both acute and chronic dysen-

[1] Read by invitation before the Nashville Academy of Medicine.

[2] Lambl: Beobacht. u. Stud. aus den Franz-Josef-Kinder-Spital, 1860.

[3] Loesch: "Massenhafte Entwickelung von Amœben in Dickdarm." Virch. Arch., Bd. lxv.

[4] Kartulis: "Zur Aetiologie der Dysenterie in Aegypten." Virchow's Archiv, 1885, Bd. cv.; "Ueber tropische Leberabscesse und ihr Verhältniss zur Dysenterie." Virchow's Archiv, 1889, Bd. cxviii.; "Zur Aetiologie der Leberabscesse. Lebende Dysenterie Amœben im Eiter der dysenterischen Leberabscesse." Centralblatt f. Bakteriologie, 1887, Bd. xi.; "Ueber weitere Verbreitungsgebiete der Dysenterie Amoeben." Centralblatt für Bakteriologie, 1890, Bd. vii.

tery. In twelve post-mortems the amœbæ were present in the ulcers in every case. He further studied microscopically the contents of twenty-two liver-abscesses, and found amœbæ present in all. Later he reports two cases originating in Athens. He also produced the disease in dogs and cats by rectal injections of the stools and both pure and contaminated cultures of the amœbæ. Massiutin[1] has, at Kiev, studied the question under Loesch's supervision. He found amœbæ, resembling in every particular those described by Kartulis, in the stools of five patients: one a case of chronic dysentery of several years' standing; the second, a man with chronic intestinal catarrh; a third, a case of typhoid fever with late diarrhœa and much mucus in the stools; the fourth and fifth were cases of diarrhœa with fluid, mucoid stools. He doubts their connection with the intestinal lesions. Hlava[2] found amœbæ in sixty cases of dysentery at Prague. He produced the disease in both cats and dogs by injecting stools containing amœbæ. Cases were also reported by Cahen,[3] of Graz, and Nasse,[4] of Berlin. Osler[5] was the first to describe the amœbæ coli in this country. He found amœbæ present in the pus of a liver-abscess complicating chronic dysentery; the amœbæ were also found in the stools. The patient, who was a physician, contracted the disease while a resident in Panama some years before. Quickly following Osler's paper reports of cases of this disease came from various parts of this country. Stengel[6] claimed to have found amœbæ in three cases of acute dysentery in Philadelphia, and a short time after Musser[7] reported four cases from the same city. Rhein,[8] also of Philadelphia, mentions that he found amœbæ in fifteen cases of dysentery, but gave no details. Dock[9] was the first to find amœbic dysentery in the South, he having reported twelve cases of both acute and chronic forms of this disease occurring in and about Galveston. In a letter, Dr. Dock informs me that before leaving Texas he saw five more cases, but has searched in vain for amœbæ at his present location in Ann Arbor, Mich. Eichberg,[10] of Cincinnati, reported a case of liver-abscess in the pus of which many amœbæ were found, but no bacteria. In the stools amœbæ were also present in moderate numbers. He also mentions that amœbæ have been found in two other cases of hepatic abscess, occurring, presumably, in Cincinnati. Lutz[11] has reported three cases from Brazil, and has added

[1] Massiutin: Centralblatt f. Bakteriologie, 1887, Bd. vi.
[2] Hlava: Centralblatt f. Bakteriologie, 1887, Bd. i.
[3] Cahen: Deutsche med. Wochenschrift, July 2, 1891.
[4] Nasse: Deutsche med. Wochenschrift, July 9, 1891.
[5] Osler: Bulletin of the Johns Hopkins Hospital, 1890, i.
[6] Stengel: Medical News, November 15, 1890.
[7] Musser: University Medical Magazine, December, 1890.
[8] Rhein: Medical News, January 9, 1892.
[9] Dock: Texas Medical Journal, April, 1891.
[10] Eichberg: Medical News, August 22, 1891.
[11] Lutz: Zur Kenntniss der Amœbenenteritis. Centralblatt f. Bakt., 1891, x.

some just criticisms of our inexact classifications of intestinal affections. Wasdin[1] reports a case of dysentery, with autopsy from Charleston, in which many amœbæ were found in the stools and intestinal contents. This case originated in Savannah, and was thought to have resulted from drinking Savannah river-water. I reported[2] three cases of chronic dysentery, with the usual symptoms, in the stools of which amœbæ were found in large numbers, differing in no essential particular from those described by Osler, Dock, and others. Fitz and Gerry[3] report a case of dysentery, with amœbæ in the stools, occurring near Boston. Preston and Ruhräh[4] report a case of amœbic dysentery from Baltimore; the patient spontaneously recovered. Stockton[5] reported a case from Buffalo in 1894. Wilson,[6] of Birmingham, recently reported four typical cases of this disease. A very interesting case of abscess of the liver, which contained amœbæ, complicating amœbic dysentery in a negro boy, has been reported by Slaughter,[7] of Theological Seminary, Virginia. Lewis[8] has recently reported a case from Baltimore.

Within the last two or three years numerous articles have been published, both in this country and abroad, on this subject. From England a case has been reported by Curnow,[9] which originated in India; others who have written on this subject are Tenoglia,[10] Wesener,[11] Kovács,[12] Epstein,[13] Posner,[14] Schuberg,[15] Kruse and Pasquale,[16] Roos,[17] Vivalvi,[18] Celli and Fiocca,[19] and Manner.[20]

By far, however, the most valuable contribution which has been made to this subject is the monograph of Councilman and Lafleur,[21] at that time of the Johns Hopkins Hospital.

As a basis for their study they reported fifteen cases (Osler's case included), with autopsies in eight. These careful observers conclude as a result of their studies, together with those of Loesch, Kartulis, Hlava, and others, that the form of dysentery especially characterized by the

[1] Wasdin: Ibid., December 5, 1891. [2] Harris: Ibid., December 3, 1892.
[3] Fitz and Gerry: Boston Medical and Surgical Journal, December 3, 1891.
[4] Preston and Ruhräh: New York Medical Journal, November 10, 1894.
[5] Stockton: International Clinics, 1894, i.
[6] Wilson: Johns Hopkins Hospital Bulletin, September and October, 1895.
[7] Slaughter: Virginia Medical Monthly, October, 1895.
[8] Lewis: Maryland Medical Journal, June 13, 1896.
[9] Curnow: Leonard's Illustrated Medical Journal, January, 1896.
[10] Tenoglia: Entéro-Colite par amœba coli. Archiv. ital. de biol., 1890, xiv.
[11] Wesener: Centralblatt f. allg. path., 1892, iii.
[12] Kovács: Beobacht. über Amœbendysenterie. Zeitschr. f. Heilk., 1892, xiii.
[13] Epstein: Prag. med. Wochenschrift, 1893.
[14] Posner: Berliner klinische Wochenshrift, 1893.
[15] Schuberg: Centralblatt f. Bakt., 1893, xiii.
[16] Kruse and Pasquale: Zeitsch. f. Hyg., 1894, xvi.
[17] Roos: Arch. f. exp. Path., 1894, xxxiii.
[18] Vivalvi: Riforma Med., 1894, No. 138.
[19] Celli and Fiocca: Centralblatt f. Bakt., 1890, Bd. ix. u. x.
[20] Manner: Wiener klinische Wochenschrift, February 20, 1896.
[21] Councilman and Lafleur: Johns Hopkins Hospital Reports, ii., Nos. 7, 8, and 9.

amœbæ in the stools "is a form of dysentery which etiologically, clinically, and anatomically should be regarded as a distinct disease."

My observations on this disease embrace the clinical study of thirty-five[1] cases—the details in three having been already published—and the post-mortem appearances in five.

Below is given a brief *résumé* of the clinical histories of these cases, the complete details being rather too tedious for publication. I desire to emphasize the fact that most of my cases were chronic. Thirty-one of my thirty-five patients with this disease were born in America, and four were of foreign birth. Of the former, eighteen were white and thirteen were negroes. Of the latter, all were Russian Jews. Of the thirty-five patients, four were under ten years of age, five from ten to twenty, five from twenty to thirty, eleven from thirty to forty, six from forty to fifty, and four from fifty to sixty at the time of the onset of the disease. Thirty of the patients contracted the disease in Atlanta Ga.; one in Decatur, Ga.; one in Athens, Ga.; one in Talladega, Ala. and two in Chattanooga, Tenn. Six lived in fairly healthy localities, while twenty-four resided in very unhealthy localities; the conditions under which five lived were not known, but with one exception were probably bad. Four denied drinking well-water at all, while thirty obtained their drinking-water from surface wells; in one case nothing was learned of the source of water-supply. In two instances two of the patients were taken simultaneously while living in the same house, and in one instance three were taken under the same circumstances. Twelve were laborers; many of the women had no occupation, and several worked at the ordinary trades. With but few exceptions the patients were from the lower walks of life. Nothing of importance developed from inquiries as to their family histories. Six had had dysentery before.

Of the thirty-five cases two were developed in March, one in April, seven in May, five in June, five in July, seven in August, four in September, and four in December. In eighteen of the cases the onset was slow, and in sixteen the disease began as a severe dysentery; the nature of the onset in one case was not determined. Twenty-three lost flesh decidedly.

After the disease becomes chronic the temperature is usually slightly elevated, especially at night, but rarely goes above 101° F. Both pulse and respiration are normal, or but little accelerated. The tongue was more or less coated in twenty-five cases; it was also often slightly reddened at the edges. Œdema was noted under the eyes in two cases, and under both eyes and around the ankles in four. In eight instances the abdomen was distended; in one it was retracted; it was tender in eighteen instances.

[1] Five of these cases were seen after the paper was read.

Sugar was not found in the urine of any of the cases, and albumin only once. Peptones, albumose, and globulin were carefully searched for in several bad cases, but none found. When the stools are excessively watery and copious, chlorides largely disappear from the urine; in one case there was total suppression of chlorides for a time. The number of stools vary from two to forty, or even more, in twenty-four hours. They vary in consistency with the varying severity of the disease. In nine cases they were very watery; in three, semi-fluid; in twenty-one, semi-solid; in one, nearly solid; and in one case undetermined. In color they vary somewhat, but in over half of the cases they were pink, reddish, or reddish-brown; rarely are they of the normal yellow color. With scarcely an exception, they were exceedingly offensive. The reaction in the first few cases was not determined, but subsequently all examined were found alkaline, except in two instances. In six analyses of the stools in two severe cases large quantities of serum albumin were found, and small amounts of peptones (Hofmeister's method) were found present in each specimen examined. Neither globulin nor albumose was found, but that small quantities were present can scarcely be doubted. Chlorides were largely present. On microscopic examination, all were found to contain, in addition to the usual undigested particles of food, pus-cells, more or less red cells, large epithelioid cells, multitudes of bacteria, and amœbæ. In six instances cercomonas were found, and in one trichomonas; in one case (a child) the eggs of oxyuris vermiculosis were found.

All complained of more or less griping and frequent desire to go to stool. Abdomen was more or less constantly painful in fourteen instances, and eight complained of backache; the backache was continuous in some of the cases, but in others intermittent—in two cases being aggravated by the recumbent posture. Four of the patients complained of nausea and vomiting, three of night-sweats, and five of fever and thirst. Obstinate hiccough was observed twice preceding death.

As complications, liver-abscess occurred twice, peritonitis twice, piles once, and appendicitis once.

Ten were treated with the ordinary astringents; two of these recovered, five died, and the result in three is unknown; one of the recoveries occurred while the patient was taking bismuth subnitrate and opium, and in the other case the result was spontaneous. Twenty-five were treated by rectal injections of solutions of hydrogen dioxide; eight recovered, four improved, two were not benefited, five died, and the result in six unknown. One recovered under injections of solutions of potassium permanganate, and another recovered while under the care of another physician. Of my thirty-five cases twelve recovered, four improved, nine died, and the result in ten is unknown.

Like the symptoms and the effects of treatment, the duration is ex-

tremely variable; one case died in eleven days; when last heard from, some had existed half as many years.

Etiology. Amœbic dysentery is a disease pre-eminently of the poor, and is almost always associated with filth, bad hygienic surroundings, and lack of proper food; hence it is that it is very rare in the country and smaller towns, but here the catarrhal form is quite frequent. Where the matter has been inquired into, it has been also found that, in a large proportion of cases, the patients had been more or less in the habit of drinking surface drainage water from wells; this was found to be true in thirty out of my thirty-five cases. As a predisposing cause, race seems to be an important factor, as out of the seventy-eight cases reported in this country (the ones I now report being included), only eighteen have occurred in the colored race. A considerable number have also occurred in foreigners. The infrequency with which it occurs in children and young adults is very remarkable—the proportion being about ten grown persons to one under twenty-one years of age. It seems to occur in males about three times as frequently as in females. Although it occasionally begins in the winter, the onset is almost invariably in the summer months—especially in May, June, July, and August. In this it differs markedly from the catarrhal form, which, in the South, is certainly more frequent in the early spring months. Attempts to connect any special form of bacterial life with this disease have heretofore proved futile, but a parasite belonging to the great group of protozoa has been found in the intestinal lesions with great uniformity. It cannot be denied, however, that positive proof of the etiologic relation of these organisms is still wanting; but when we consider that amœbæ, the descriptions of which, from all sources, differ very slightly, have been found constantly present in the stools of a common, chronic, and very fatal form of dysentery, in various parts of Europe, Asia, Africa, and in both North and South America, and that they occur in the stools of other diseases certainly very rarely—which might be easily accounted for by supposing some complicating amœbic pathological process—it is strong presumptive evidence that they play an important rôle in the production of the pathologic changes which occur in this disease; when we further consider that competent observers have always succeeded in demonstrating these organisms in and around the ulcers which occur in the colon, and that one observer, by the rectal injection of cultures of the amœbæ, has produced inflammation and erosion of the mucous membrane of the large intestine of lower animals, the evidence becomes even stronger that they are the active causative agents in this affection. Further, large numbers of amœbæ—sometimes occurring in pure cultures—are found in abscesses of the liver and lungs, complicating amœbic dysentery. Flexner,[1] in

[1] Flexner: Johns Hopkins Bulletin, September, 1892.

a very interesting article, describes amœbæ occurring in the pus from an abscess of the lower jaw, which he regards as "allied species if not identical" with the amœbæ dysenterie.

In collecting the feces for examination for the amœbæ, some precautions are necessary to prevent the death of these organisms before the examination is made, as, when dead, they resemble very much the large epithelioid cells which often occur so abundantly in dysenteric stools; the stools should be received directly in a perfectly clean vessel, which in cold weather may be warmed, and should contain no water or other fluids, urine being especially damaging. The amœbæ are from twelve to thirty-six micromillimetres in diameter. Generally the body of an amœba can be distinguished as being made up of an outer, clear homogeneous substance or ectosarc, and an inner, highly refractive mass or endosarc; within the latter are found foreign bodies, such as bacteria, granular detritus, bodies which seem to be red blood-corpuscles, and one or more large vacuoles, and great numbers of smaller ones, giving the appearance of granulation; they are frequently found, however, with few or no vacuoles of any size. The impression has gradually grown upon me that amœbæ in each case bear a general resemblance to each other in respect to the number and size of their vacuoles, or at least it is true for comparatively lengthy periods, if not throughout the entire course of the disease. Within the vacuoles, bodies in active Brownian movement may be sometimes seen. In consistency the ectosarc is semi-solid, allowing the amœbæ to push readily through the granular débris found in the feces, but the endosarc must be quite liquid, for on one occasion, within the body of one of these organisms, which was almost entirely free of vacuoles, an actively moving bacillus was seen. When dead, before disintegration begins and when not active, the organisms are generally round or nearly so, but occasional exceptions are seen. When in motion almost every conceivable form is assumed, barring that the irregularities of the amœbæ body and the processes sent out are invariably rounded. Progression of the amœbæ may be accomplished by a steady crawling movement, or, what is more usual, pseudopodia are sent out, followed by the contents of the body flowing into them, which causes a rapid increase in the size of the pseudopodia and continues until the entire body changes place. While living, the amœbæ are not affected by currents under the cover-glass, but when dead they drift like other inanimate particles of matter. The cell contents frequently change place without perceptible movement of the ectosarc. While moving, a collection of fecal detritus is often attached to the posterior portions of the amœbæ, and frequently I have been unable to make out the exact point of separation; indeed, it has seemed to me, at times, that there was direct continuation of one into the other. If this is true it may explain what has always puzzled me—*i. e.*, how do the amœbæ take in their

food and the various cellular elements which are almost always found in their bodies? In this connection, the observations of Cienkowski[1] Duncan,[2] Leidy,[3] and Cunningham[4] on this subject are of great interest. As far as my observations go, pseudopodia are never sent out at the point at which these masses are attached to the amœbæ. The bodies of the amœbæ usually have a faint green or bluish-green color. A nucleus may sometimes be distinguished in them, but by no means always; it is usually surrounded by a white rim; they sometimes change their form slightly. No contractile vesicle was ever seen.

The length of time that they live after being passed varies considerably in each case; amœbæ from Case I.[5] often remained alive and active for twenty-four hours in a room where the temperature fell almost to the freezing-point during the night; this, however, I regard as being very exceptional. As a general rule, they die in from two to six hours after expulsion from the intestine. Writers on this subject have claimed that temperature exerted a marked influence on the length of time which they would live, but, while believing that a temperature about that of the body is most favorable to them, I have so far seen nothing to convince me of the truth of the above conclusion. I have often noticed that, from the same case, amœbæ died about as soon at the temperature of the room as when kept at 37° C. in an incubator. My experiments to determine the toxic effects of various agents on amœbæ have been confined largely to Case I., in which, as above indicated, the amœbæ seemed unusually tenacious of life. The organisms in this case were not killed in any reasonable length of time by lowering the temperature of the fluid in which they were contained to 0° C.; neither were they seemingly at all affected by saturated solutions of quinine sulphate or boric acid—both of which have been recommended for the purpose. A 1:300 aqueous solution of quinine bisulphate invariably killed the amœbæ within ten minutes. They are killed by weak solutions of hydrogen dioxide, potassium permanganate, and toluidine blue; they are also killed by acids, even when very dilute, and for this reason, largely, the urine acts in a similar manner, but its toxic effect is not entirely due to its acidity, as I have found that after being neutralized the amœbæ do not live very long in it. Agents, rectal injections of which were likely to prove harmful to the individual, were not experimented with.

As shown by me,[6] the amœbæ sometimes reproduce after being ex-

[1] Cienkowski: Archiv f. mikrosk. Anatomie, 1865, Bd. i. S. 203.
[2] Duncan: Popular Science Review, 1877, p. 217.
[3] Leidy: Fresh-water Rhizopods of North America, p. 27.
[4] Cunningham: Quarterly Journal of Microscopic Science, 1881.
[5] For convenience, the cases are referred to in this paper in the order in which they were seen.
[6] Harris: Medical News, November 24, 1894.

pelled from the intestine; multiplication takes place by direct division. Attempts to cultivate them in straw infusions, as recommended by Kartulis,[1] have always been unsuccessful.

For a long time I was unable to find a stain which would color the amœbæ in the fresh state, as they showed not the slightest tendency to take any of the ordinary dyes. Some progress was made by adding to the fresh cover-slip preparations of fecal matter a drop of an aqueous solution of acid fuchsin, which has the effect of coloring deeply all objects ordinarily found in the stools except such crystalline bodies as might be present and the amœbæ. After having discovered the great affinity that toluidine blue displayed for the fixed amœbæ, at the first opportunity I added a drop of watery solution of this substance to a small particle of the feces, and examined it immediately under the microscope; here, as in the tissues, the endosarc of the amœbæ was always intensely stained at once; the ectosarc only becoming so after the lapse of several minutes. In fresh preparations the ecto- and endosarc are beautifully differentiated. The amœbæ seemed to be instantly killed, and often their natural forms were beautifully preserved. The cover-slips, after being washed in water and mounted in Farrant's medium, may be preserved for many months, but after a time the preparations completely fade. It is evident that in addition to staining the dye also acts as a fixative. Weak solutions of the stain should be used, or the cover-slip should be washed in water before mounting. In staining the feces by this method, frequently small, vacuolated bodies were found which take the stain as do the amœbæ; these bodies are about the size of leucocytes, but differ from them in many particulars.

Attempts were made to stain the amœbæ in cover-slip preparations of the feces by first placing both the dried and moist cover-slips in various hardening and fixing solutions. Alcohol, both weak and strong, Müller's solution, strong solutions of potassium permanganate, 2 per cent. osmic acid solutions, strong and weak solutions of chromic acid, Hermann's solution, Flemming's strong solution, and Heidenhain's mercury bichloride salt solution, with a little acetic acid added, were all tried, with but very indifferent results—the last three, however, giving the best. By none of the methods were they sufficiently well preserved to present more than a very faint resemblance to these organisms when alive; none of the above agents killed rapidly enough to prevent enormous shrinkage and the bodies of the amœbæ from losing their more or less characteristic irregular form and becoming rounded. Scarcely better results were obtained from sections of fecal masses, which were treated with the above hardening agents, embedded, cut, and stained.

[1] Kartulis: "Einiges über die Pathogenese der Dysenterieamoeben." Centralblatt f. Bakteriologie, 1891, Bd. ix.

Strangely enough, and this fact is mentioned by Councilman, the amœbæ are much better preserved by hardening agents in the tissues than in the feces; but even here, in the most favorable cases, they shrink considerably, and generally lose largely their characteristic vacuolation; the only explanation of this fact which has occurred to me as being at all likely is that the solutions do not reach the amœbæ at first in so concentrated a form as when merely the feces are used; but this, it must be admitted, is a mere assumption without any real proofs, and which even seems unlikely when it is remembered that the results are practically the same whether great or small masses of feces are used.

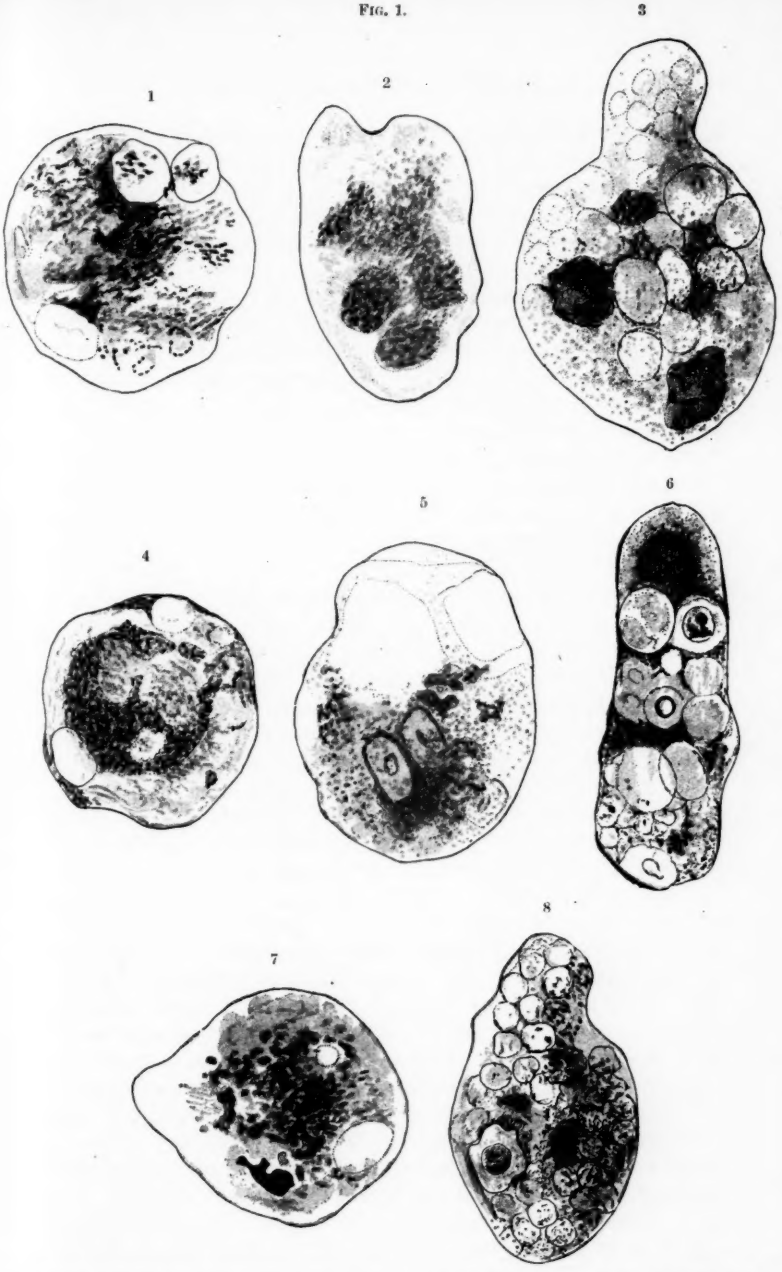
The amœbæ were well preserved in the tissues when acidified Heidenhain's mercury bichloride salt solution, Hermann's solution, Flemming's solution, Müller's solution—made very strong and placed in the incubator for three days, as recommended by Councilman—and alcohol were used as hardening agents—the first three, as in the case of the feces, giving the best results. A large number of staining reagents were used on the tissues hardened in each of the above solutions, with varying results. Councilman states that the amœbæ are probably most easily distinguished after hardening in alcohol and staining with Loeffler's methylene blue; I would add that results quite as good, if not better, are obtained with this stain by fixing in corrosive sublimate, instead of hardening in alcohol. Now, while the amœbæ are brought out very prominently by the above stain, it is by no means to be recommended for studying the delicate internal structure of these organisms, as the entire endosarc seems to take up nearly the same amount of the stain, and their bodies become so dark that the vacuoles are no longer apparent, and the nuclei are not distinguishable with certainty. The iron hæmatoxylin of Buetschli gives much the same general effect, the only difference being that they stain darker. After fixing in any of the above reagents the amœbæ are especially well stained by blue solutions of hæmatoxylin, but the same objection applies in this case as was mentioned in connection with methylene blue, namely, that the amœbæ were so intensely colored that the internal structures could not be observed; fairly good results were obtained by first staining with hæmatoxylin and after with eosin, benzopurpurin, or picric acid, the last three, however, always washing out the first rapidly. After hardening in Heidenhain's solution, they stain brilliantly in the carmines, but not quite so dark as the tissues; very beautiful and delicate stains were obtained with weak alum carmine containing a trace of osmic acid. The amœbæ stain fairly well by Bizzozero's modifications of Gram's gentian violet aniline-water method; staining first with gentian violet, and following with eosin, gives much the same effect, but I fancy somewhat better than hæmatoxylin and eosin; the nuclei are beautifully stained in this way. Nigrosin stains the bodies of the amœbæ well, but the nuclei are

not brought out. The Ehrlich-Biondi-Heidenhain stain produces delicate but very faint results. I have never seen the nucleus stained by the methylene green of this combination, but strangely, the ectosarcs of the amœbæ stain faintly with this color; this stain works best with corrosive sublimate objects. Victoria blue stains the nuclei faint blue; no other part of the organism is at all colored, however. The bodies of the amœbæ stain yellow by Van Giesens' method; the nuclei are faintly stained. Very fine results were obtained by staining with carbol-fuchsin, the bodies of the amœbæ stain a beautiful purplish red, the borders and centres of the nuclei being a dark purple; the stain is perhaps somewhat more decided after hardening in osmic acid solutions. Safranin stains the endosarc a reddish color, some parts looking, however, quite dark; the irregular nuclei are stained dark in the centre and at their margins, and the ectosarc faintly red; perhaps no method shows the division of the amœbæ into endo- and ectosarc so well as this one. Equally good, if not better, results in some particulars are obtained by first staining lightly with hæmatoxylin and after with safranin, according to the method of Rabl. Flemming's orange method gives very fine results—the nuclei being very distinct and the protoplasm darker than when safranin is used alone. It is noteworthy, as is the case with the tissues, that all safranin stains are fainter and less precise when the amœbæ have been hardened with corrosive sublimate than when hardened in osmic solutions. The bodies of the amœbæ are slightly and the nuclei very decidedly stained by Weigert's fibrin stain.

Although very fine stains may be obtained by some of the above methods, decidedly the best results, so far as the internal structure of the organism is concerned, have been obtained by hardening in corrosive sublimate and staining in Heidenhain's iron alum hæmatoxylin; the method gives a dark, very transparent, and highly differentiated coloration, but permits the vacuoles to be seen as in no other stain; the nuclei are irregularldy stained, being darkest on the periphery and at their centres. Furthermore, the amœbæ, when stained by this method, can be detected in the tissues almost as readily as if stained by methylene blue, and are much more easily distinguished from objects resembling them. When the above method is used the amœbæ very frequently closely resemble those in a living state, the only difference being that there is always more or less shrinkage. (Fig. 1.)

Having observed the fact, as already mentioned, that the protoplasm of the amœbæ exhibited marked affinity for the blue solutions of hæmatoxylin and methylene-blue, it occurred to me that perhaps the endosarc of the amœbæ was composed of a mucus-like substance; I therefore stained some specimens of intestine containing these organisms, which had been fixed in corrosive sublimate, with a weak watery solution of toluidin blue, and found the amœbæ were much more deeply

FIG. 1.



Amœbæ and a cell from the margin of a liver abscess. Toluidin-blue and eosin; Zeiss $^1/_{12}$ oil immersion, Oc.C. Nos. 1, 5, and 8 were fixed in Hermann's solution; Nos. 2, 3, 4, 6, and 7 were fixed in corrosive sublimate. No. 1 was stained by Flemming's safranin, gentian-violet, and orange stain; No. 5 by Robb's hæmatoxylin-safranin method; No. 8, which is a degenerate liver cell, and is put here for the purpose of comparison, was stained with carbol-fuchsin. Nos. 2 and 4 were stained by Loeffler's methylene blue. Nos. 3, 6, and 7 were stained by Heidenhain's iron-hæmatoxylin method.

stained than the tissues. After experimenting further it was observed that a beautiful contrast stain could be produced by staining first in eosin or benzopurpurin, and after, for twenty or thirty minutes, in a weak solution of toluidin blue; the excess of the latter stain is washed out with alcohol, which usually takes three or four minutes, and the section is then cleared in cedar oil or xylol, it having been found that other clearing agents more or less injured the stain. If the sections are washed in alcohol for only a short time the amœbæ are stained a very solid dark blue, in which their nuclei can be seen stained of even a darker color, but if the washing is prolonged their bodies are not so dark and have a reddish tinge, the vacuoles becoming quite distinct, and the outer portions of the ectosarc retaining the pigment very slightly; the tissues are a purplish red, while the nuclei stain very nearly the same color as the amœbæ. If the eosin is used after the toluidin blue this stain is washed out of the amœbæ. By this method it is possible to examine with a low power a large section for these organisms in a few moments. It is certainly by far the most beautiful stain yet discovered for the amœbæ; indeed, it would be hardly possible to devise a better. It seems quite likely that the endosarc is made up of a substance greatly resembling mucin, and the inference is not without justification that the endosarc of other rhizopods and other members of the protozoa may be also composed of a like material, and may give the same reactions with toluidin blue. It kills cercomonas quickly, but scarcely stains them at all. Although somewhat out of place, I would call attention to the fact that the combination of eosin and tuloidin blue is a most excellent method for general histologic work, and compares favorably with the results obtained by staining with eosin and hæmotoxylin or gentian violet. Unfortunately, the stain gives differential effects only with corrosive sublimate or alcoholic objects. Other mucin stains, as thionin, muchæmatein, and mucicarmine, were tried, with excellent results, but none gave such fine effects as the toluidin blue; the results with thionin were especially good, but the stain cannot be used with eosin and the like, as it will not replace these acid dyes in the amœbæ. In this connection it may be noted that hyaline cartilage, mastzellen, and the jelly of Wharton give the same reaction with basic dyes as mucin does.

From the above, it will be seen, as should have been expected, that the bodies of the amœbæ have much more affinity for plasmatic than for nuclear stains, and that the latter are always washed out by the former. Within the bodies of the amœbæ may be generally seen some granular detritus, the result, no doubt, of cellular disintegration; but I have found it extremely difficult, after staining, to recognize with any certainty included cells. Neither have I in a single instance been able to make out the radiate-like structures described by Councilman as sometimes occurring in the protoplasm of these organisms; the fluid nature

of the endosarc would seem to make very improbable the existence of these structures during life, and when it is remembered that they were found only in tissues hardened by Flemming's solution, which contains a large proportion of chromic acid, the possibility is strongly suggested that they may be artefacts. I have never seen fat in the interior of the amœbæ.

The question as to how the amœbæ gain access to the system is one of great interest, and a perfectly satisfactory answer has, as yet, not been given. In all of the cases reported by Councilman and Lafleur the patients were drinking well-water at the time of the onset of the disease, and with but three exceptions—and even here it is a matter of doubt in two of these—the same is true of all my cases. Of great interest in this connection is the case reported by Fitz and Gerry, as these observers demonstrated the presence of amœbæ, in most respects resembling those found in the feces, in a cistern from which the patient constantly used water. A careful examination of the water used by patients in Cases XII. and XXIV. failed to reveal the presence of amœbæ; neither were they found in the mud from the bottom of the wells or scrapings from the well-bucket.

Pathological Anatomy. On opening the abdominal cavity in Case IV. several localized sacs of pus were found; and from an ulcer which .penetrated through the entire thickness of the gut there was a general peritonitis; this was also found in Case XIV. There was a considerable amount of pus found in the peritoneal cavity, in both cases of a whitish-yellow color. In Case IV. there were adhesions ot the large intestine to all contiguous objects; the gut was much distorted, being drawn in first one direction and then another, as was also the small intestine, which was fastened to it wherever they came in contact. Most of the adhesions were old and very firm. The large intestine was at some points enormously thickened; at others so thin that it was impossible to avoid many tears during its removal. In all cases the intestinal contents consisted of a mass of foul-smelling semi-solid fecal matter, of a dark-brown color, containing particles of undigested food, blood, and much mucus. Amœbæ in the feces were sought for only in Cases XIV. and XXII., and were found in both. The characteristic lesions in this disease are the ulcers of the large intestine. These ulcers may extend almost continuously from the rectum to the ileo-cæcal valve, or may be confined to one or more limited areas. Rarely, as in Case XIV., the ulceration may extend into the small intestine. A novel feature, which I have not seen mentioned in reports of others, is that the appendix was also involved in this case. This appendage was found surrounded by pus, which contained amœbæ, was much enlarged, and was bound, for its full length, to the small intestine by firm adhesions. At one point the walls had been completely destroyed,

thus establishing a communication between its cavity and that of the surrounding abscess. On section, the appendix was found filled with thick whitish pus, in which many amœbæ were found. The ulcers are found in greater number on the lower and anterior part of the gut. It was especially noted, in all cases, at the points where the mesocolon was attached, and where the guts were fastened to the sides of the abdominal walls, that ulceration was practically absent. The ulcers are sometimes round or irregularly shaped, but are generally oblong, and lie transverse to the long axis of the colon; a form of the latter very frequently seen begins an inch or an inch and a half from the attached part of the gut, with a rounded extremity, and tapering somewhat, becoming lost near the point just named in several irregular continuations which follow the depressions between the numerous rugæ in this situation. They vary very much in size—some being no larger than the head of a pin, while others are sometimes as much as two inches in diameter. In the larger ulcers the muscular coats are frequently dissected up for some distance. These masses of muscular tissue sooner or later become detached, and are found in the stools as sloughs. In none of my cases was that honeycombing of the tissues observed that Councilman and others have described, but it was usually the case that the edges of the ulcers were somewhat undermined.

When sections of the intestine are examined with the microscope, the walls are usually found normal at a short distance from the edge of the ulcer. Approaching more closely, either one or the other of two changes may be first observed: either the submucosa shows marked changes, with the mucous membrane above practically normal, or the mucosa may be eroded and no abnormality beneath.

In the former, which is by far the more frequent, and which may be considered the typical intestinal lesion of this disease, changes in the submucosa may be traced in advance of the surface ulceration for quite a distance, thus undermining the comparatively healthy mucosa above. The appearance of these ulcers when fresh is entirely characteristic. A short distance from the point where the ulceration begins the upper portion of the glands of the mucosa are slightly but irregularly eroded. From this point to the edge of the ulcer the cells of the interglandular reticulum are usually slightly increased in number. The most careful search with high powers failed, however, to show any evidence of mitosis. At a short distance from the edge of the ulceration amœbæ are not found in the mucosa, but rarely one or two of the parasites may be seen either within or between the glands just at the margin. More or less marked fatty degeneration of the cells of the mucosa occur, which is always most intense at the edge of the ulcer. The glands contain great quantities of mucus, which become especially marked at the edge of the ulcer. Often from some part

of the margin of the ulcerated mucosa a band—usually at first small—of irregular and ill-formed mucous tissue passes off, which generally becomes larger, and may extend for considerable distances over the ulcerated submucosa, or may be reflected back over the healthy mucous surface. After passing back over the healthy mucosa these bands are often reflected back over themselves—which may be repeated several times. The large bands sometimes divide into two small ones. When growing over the ulcer they seem sometimes to become attached to its surface, but this is very rare in my cases. I have never seen epithelial cells lining the ulcers, as described by Councilman and others. Amœbæ—so rare in the healthy mucosa—are quite frequently present in these outgrowths just described, but appear to exert no influence on the growth or breaking down of these bodies. The bloodvessels of the mucosa are sometimes found greatly distended with blood; but, in my experience, this is always accomplished by a high degree of bacterial infection. At the centre of the ulcers the muscularis mucosæ is always broken down, but it suffers much less than the tissue beneath, and may be often seen to extend in a comparatively healthy condition beyond the mucosa, thus covering the broken-down tissue beneath. The above, no doubt, partially explains why the breaking down of the mucosa does not keep pace with that of the submucosa. That part of the muscularis mucosæ which hangs over the ulcer is swollen, and toward the free end the fibres are granular, and their nuclei are for the most part gone, or entirely absent. That part at the sides of the ulcer, and extending back for some distance, is swollen, and the nuclei are increased in number. Where the ulceration is recent the altered submucosa forms the base of the ulcer. The breaking down here is always irregular, giving the ulcer a ragged appearance. Often almost detached shreds of this necrotic tissue lie on the free surfaces of the ulcers. Amœbæ may sometimes be seen lying on or between these shreds, but they are rarely so numerous as deeper down and along the outer edges of the diseased tissue. The surface of the ulcer is more or less covered with a granular detritus mixed with round cells, and a few epithelial and pus cells. These are not regularly distributed, but are often collected in small masses. Bacteria were also present, but never to the extent that would be supposed. The most common organism is one that morphologically resembles and stains like the colon bacillus. The most marked and characteristic changes are always present in the submucosa. The first change is a great swelling of the tissues. Immediately following there is an infiltration with small round cells, and sometimes a few polynuclear leucocytes are also seen, but these are rare. Frequently present also are large, vacuolated, mononuclear cells, which, on account of their size and general appearance, closely resemble amœbæ. They are scattered through the tissues everywhere, but are most frequent where the disease-process is most actively advancing, and are at these points

often very numerous. They seem to be connective-tissue cells which have absorbed liquids. This assumption is supported by the fact that the connective-tissue cells swell along with the tissues, and may be even seen in all stages of the transformation. They stain a light red with a blue nucleus with the toluidin blue eosin stain. At the margin of the ulcers the protoplasm of these cells often undergoes a sort of parenchymatous degeneration, and at the same time the nucleus swells and becomes indistinct. These cells all stain poorly. Mastzellen are diminished in number in the immediate vicinity of the ulcers. Immediately following the swelling and infiltration of the submucosa the more delicate connective tissues soften and break down, and, usually, in the order named—the veins, arteries, and large bands of fibrous tissue. As soon as changes begin in the submucosa the connective tissues become greatly swollen, quickly lose the power of retaining stains, as when normal, soften, and break down. The large bands, especially, show a tendency to undergo a sort of hyaline transformation at or very near the free surface of the ulcer. I have never found this hyaline change very extensive, nor have I observed it forming a reticulum. Before undergoing the change or breaking down, the large bands of fibrous tissue, like the smaller, become pale and take stains poorly. The elastic tissue does not suffer equally with the collagenous fibres, but remains intact and retains its staining peculiarities long after the surrounding tissues have broken down and disappeared. Perfect bands of this tissue are thus often found lying in masses of granular débris at the very edge of the ulcerated surface. Scattered through the tissues everywhere amœbæ are found, but, as a rule, are most numerous near the points where the most active changes are occurring. Very rarely a single amœba may be found somewhat in advance of the pathological process, but the great mass of these organisms follow, rather than precede, the characteristic changes. They lie almost always in the lymph spaces and in the lymph sinuses, which are numerous in the submucosa. They do, however, occur in the tissues, where a clear zone may almost always be seen around them, which probably results from contraction caused by the fixing agent. No direct local action of the amœbæ can, as a rule, be observed. Amœbæ are also frequently found in the perivascular lymph channels. When amœbæ are present proliferation of the endothelial cells which line the lymph sinuses is often seen. The lymphatics may be considerably, and are often enormously, dilated. Small amounts of fibrin are sometimes found in these sinuses. Masses of fibrin are often found in the tissues just below the ulceration. The walls of the veins are early infiltrated with round cells, quickly followed by softening and complete disorganization. This, no doubt, explains largely the constancy of hemorrhage in this disease. As a rule, the veins contain no more blood than normal. Thrombosis of the veins is not infrequent; but, very strangely,

it seems independent of the presence of amœbæ within the vessels, where they are quite frequently observed. They may occasionally be seen in the act of penetrating the walls of the veins—part of their bodies being within and part without the lumen of these vessels. In like manner the walls of the arteries are infiltrated, and there is evidence of endarteritis, more or less pronounced, as the ulcerative process is slow or rapid. In chronic cases the lumen of the arteries may be entirely occluded by this process. The walls of the vessels sometimes undergo the same hyaline change which occurs in the connective tissue, generally beginning on the side nearest the ulceration. Before the ulceration reaches the muscular layer the nuclei of the fibres swell up, become more oval in shape, and greatly increase in number. After the ulcerative process passes through the submucosa the inner muscular layer, for a time, checks the further progress of the disease. The amœbæ, however, soon force their way into the intermuscular septa, and here cause the same changes which are produced in the connective tissues above. Simultaneously the connective tissue envelope of the muscular layer, which lies next the submucosa, breaks down, and the amœbæ penetrate between the individual fibres. In none of the other tissues are the local effects of the amœbæ more pronounced, and in none can the changes produced be so accurately observed. As with the submucosa, the swelling is pronounced, but with the difference here that the amœbæ precede or keep pace with this condition. The mechanical effect of these organisms on the fibres is always at once obvious. The cement substance softens and gives way, and the amœbæ pass onward between the fibres, pushing them to either side. The fibres swell somewhat, and soon soften, become granular and indistinct, lose their nuclei, and finally entirely break down. The bloodvessels of the muscular layer suffer the same changes which occur in those of the submucosa. As soon as the amœbæ force their way through the circular muscular layer, they pass outward in all directions, and immediately there follow the same swelling, infiltration, and necrosis of the tissues between the two muscular coats, which, under similar circumstances, occur in the submucosa. In the same manner the bloodvessels are destroyed, and, in addition to the already existing disease-producing causes, there follow those changes which result from cutting off the blood-supply. In this way quite extensive sloughs may occur. In the longitudinal muscular coat there is found an exact repetition of the changes which are produced in the circular layer. While the amœbæ are still in the muscular coats, and before any of them pass entirely through, the bloodvessels of the subperitoneal coat become engorged with blood, there are small round cell infiltration, swelling of the tissues, and, in fact, all the phenomena which precede the amœbæ in the submucosa, except that here there is always considerable connective tissue hyperplasia. After the amœbæ reach this coat there may be ob-

served the same necrosis of the tissues and breaking down which occurs in the submucosa. This process may extend outward, and a portion of the peritoneum, becoming necrotic, sloughs out, causing perforation into the peritoneal cavity. More usually, however, the peritoneum thickens more rapidly than the necrotic process advances, which often continues until an enormous thickening of this coat results. Fibrin formation is especially frequent on the peritoneum and in the subserous coat. It is a notable fact that the hyperplastic tissue which is produced in this disease is much more resistant to the disease-producing cause than are the original tissues. The question as to how the amœbæ reach the submucosa is one of great interest. Councilman thinks that they penetrate the mucosa and muscularis mucosæ, and then produce the highly characteristic lesions which always accompany their presence. This certainly seems the only probable hypothesis, if we reject the possibility that the disease may begin as a catarrhal inflammation, and that later the ulcers become secondarily infected with the amœbæ—a supposition that the microscopical anatomy of the ulcers seems at once to refute. On the other hand, it should not be forgotten that the amœbæ penetrate mucous and muscular tissue only with great difficulty, and that they are rarely found in these tissues—especially in the former. In chronic cases, adhesions of the gut to neighboring objects is quite frequent, and, as a result, great distortion sooner or later follows. When these adhesions connect with the small intestine, deformities, almost as great, occur also in this. In some cases the ulcers do not follow throughout a typical course. In Case VI., particularly, were found many ulcers which extended only a short distance below the mucous membrane, and in which there was an absence of most of the phenomena above enumerated. The fibrous tissue of the submucosa in these ulcers was considerably thickened, and bore a striking resemblance to the new-formed fibrous tissue of the peritoneum already referred to. Many amœbæ were found in this tissue, but seemed to exert little, if any unfavorable action upon it. Amœbæ were found in the walls of the ulcers of the ileum and appendix in Case XIV. (Figs. 2 and 3.)

The other form of ulceration, spoken of above, was found only in Case XIV., but seemed quite as frequent as the more typical form in this instance. These ulcers increased in size by gradual softening and breaking down at the surface—never by necrosis and sloughing of the underlying tissues. They sometimes do not extend deeper than half through the mucosa, but generally reach into the submucosa, and rarely extend to the circular muscular layer, but never deeper. They are characterized by the fact that they are more properly a lesion of the mucosa, any deeper ulceration being apparently secondary, and that there is never that swelling and infiltration of the submucosa in advance of the breaking down which characterizes the ulcers already spoken of. Further-

FIG. 3.

Section of intestine just below ulceration. Toluidin-blue and eosin. Beck ¼, Oc. 3. In upper portion of the field a large vein is seen; the wall of the vessel which is nearest the ulceration is being infiltrated with small cells and is breaking down; both red and white cells and amœbæ are seen within the lumen of the vessel. In the lower portion of the field many amœbæ are seen, some in the tissues, and others in the lymph spaces and lymph channel.

more, *they never contain amœbæ.* That abrupt breaking down of the mucosa at the margin of the ulcers which is so common in the other variety is here extremely rare, and never is there that undermining of the mucosa or that luxuriant proliferation of mucous tissue which is almost invariable in the other case. On the other hand, the mucous surface is more or less eroded for some distance from the deepest part of the ulcers, and the glandular epithelium beneath is usually entirely gone or lying in loose masses in the cavities they formerly lined. The blood-vessels are, moreover, more completely distended with blood than is usual in the other variety of ulceration. When the breaking down

FIG. 2.

Edge of intestinal ulcer. Toluidin-blue and eosin. Beck 1 inch, Oc. 3. *a*, mucous coat, which projects over ulcer at *f*; *b*, submucosa; *c*, circular layer of muscle fibres; *d*, tissues of mesocolon; *e*, amœbæ in dilated lymph spaces.

extends into the submucosa there is comparatively little swelling of the tissues, but the small round-cell infiltration is more pronounced than is the case with the ulcers first described. The large, swollen connective-tissue cells, which are so numerous in the other ulcers, are never here observed. The chronic nature of the process in these cases is shown by the fact that there is a decided increase of the fibrous tissue in the neighborhood of the ulcers. The walls of the bloodvessels suffer but little change, and break down only when they are actually reached by the ulceration. The vessels are usually distended with blood. Hyaline changes were never found, either in the tissues or in the walls of the bloodvessels. The lymph vessels are not dilated, nor is there any

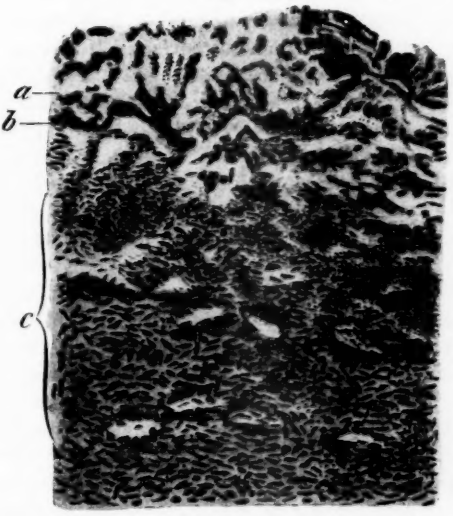
marked change in their walls in advance of the general breaking down of the tissues. After the ulceration reaches the circular muscular coat the number of nuclei in the fibres is sometimes increased, but is never so decided as in the former variety. Under like conditions, the intermuscular septa may become thickened.

The lymphatic glands of the large intestine in Case XXII. were found somewhat swollen, but were not hard. On section, they presented the usual appearance. Pieces were fixed in corrosive sublimate and Hermann's solutions. Sections were stained by the same methods which were used in staining sections of the intestine. On microscopic examination the lymph sinuses were found considerably dilated, and contained numerous large, swollen cells which almost always contained one, and more frequently numerous vacuoles. These cells usually have only one nucleus, but there are frequent exceptions. Mitosis can sometimes be observed. These cells have occasionally no nuclei, but in these cases the protoplasm takes the stain poorly, and the entire cell is evidently undergoing degeneration. The protoplasm of these cells is acidophilous, and hence shows a red coloration with a dark nucleus when stained by the eosin toluidin blue method. They resemble in every particular the swollen connective-tissue cells which are so numerous in the vicinity of the ulcers, and, like them, greatly resemble amœbæ, especially when stained by some methods. They contain no fat. The lymphocytes of the glands are probably somewhat increased in number. No change of the reticulum or of the bloodvessels is observed. The glands in Case XXXV. were also swollen. They showed a decided increase of lymphocytes and a few of the above-mentioned cells.

In Case XIV. there was an hepatic abscess. The liver was pale in color and was considerably enlarged. It was attached firmly to the diaphragm wherever in contact with it. At one point the ulceration had extended through the capsule of the liver, and almost through the diaphragm. On section, the right lobe was found to be almost completely destroyed, the capsule constituting at many points the only wall of the abscess. One abscess of considerable size and many small ones were found scattered through the left lobe. The inner walls of all of these abscesses are very irregular, and in the larger ones present an extremely ragged appearance. It is a curious fact that these abscesses, as in the present instance, show a much greater tendency to rupture into the lung than into the intestines or peritoneal cavity. The contents of the abscesses were numerous shreds of partially disintegrated tissue, and a creamy, reddish-yellow fluid which much resembled pus. Under the microscope this fluid is found to consist of a mass of granular detritus, a few pus-cells, and numerous amœbæ. Pieces of the liver tissue were hardened and stained by the methods already described. In the edges of sections which had been taken from the walls of the abscesses and in small

abscesses, which could be examined entire, the pus-like fluid which they contained was found to consist largely of a granular mass, in which were a few pus-cells, some nearly destroyed liver-cells, here and there shreds of masses of fibrin, some fibrinous remains of the connective tissue of the liver, which often contained bloodvessels and bile-ducts, many amœbæ, and, at some points, multitudes of cocci. This fluid also contained numerous nuclei in a state of partial disintegration, and occasionally small masses of fat. (Fig. 4.) The most interesting of these constitu-

FIG. 4.



Section of edge of a liver abscess. Toluidin-blue and eosin. Beck 1 inch, Oc. 3. *a*, broken-down mass which constitutes the contents of the abscess; *b*, the abscess wall; *c*, the breaking down liver cells in the vicinity.

ents are the masses of connective tissues. They often lie at considerable distances from the abscess-wall, and frequently appear to have no connection with it. The degree of vitality which they show under these circumstances is certainly very remarkable. Around their edges the tissues are always softened, their nuclei are swollen, partially broken down, and do not take the stain, and all stages of disintegration may be observed. The central portions of these masses are comparatively little changed, but the tissues are somewhat cloudy and are swollen to a slight extent, and their nuclei are increased in number. Around the margins of these masses is a favorite haunt of the amœbæ.

From the foregoing it will be at once seen that the contents of these abscesses is not pus—indeed, it much more closely resembles the cheesy

masses of tuberculosis. The walls of the abscesses differ somewhat, depending on their size and age. In the small and more recent ones it cannot be said that a wall exists, for the abscess contents gradually, and in a most irregular manner, merge imperceptibly into the surrounding half-degenerate liver-cells. In the larger abscesses the walls—while extremely irregular—are, as a rule, comparatively well defined, and consist of a mass of spindle-shaped or rounder bodies, which are evidently partially broken-down liver-cells. Frequently masses of fibrin constitute a part of the walls, and, interlacing between these cells, form a sort of reticulum. Amœbæ are often found along the inner border of the walls, and sometimes within them, but never penetrate farther than a few micromillimetres from the surface. Numerous colonies of bacilli resembling those found in the intestinal ulcers, and corresponding in their staining properties and their appearance to the colon bacilli, were found along the inner border within and beyond the walls. It would be difficult to conceive of a greater destruction of the liver-cells than occurs in the vicinity of the abscesses, and, indeed, most marked changes of the liver substance can be observed even at points most remote from these lesions. The liver-cells become granular, break up, and are detached from their capillaries. They also become much narrower than normal. Occurring usually in groups, are found very refractive granular cells, which evidently result from necrosis of the liver-cells. These bodies are almost always rounded or oval in shape, contain usually one or more well-defined nuclei, and generally many vacuoles, some of which are composed of fat, as may be seen in tissues fixed in osmic solutions. They are usually from eight to twenty micromillimetres in diameter, and might be easily mistaken for amœbæ. They are stained red with dark nuclei with the toluidin blue eosin method. Closely related to the above are very peculiar long, often irregular, somewhat spindle-shaped bodies, which take the place and result from the necrosis of liver-cells. Indeed, the tissue for some distance around the abscesses is composed almost entirely of these cells. They are highly refractive granular bodies, which contain vacuoles, and sometimes fat, and, occasionally, one is found containing a nucleus. They are from ten to fifty micromillimetres in length, and from three to ten in width. The peculiar variety of degeneration of the liver-cells just spoken of seems almost characteristic of this disease, as I have rarely seen it around abscesses which contained no amœbæ, and, at the most, I have seen only a few broken-down cells which somewhat resemble those found in the walls of amœbic abscesses. (Fig. 5.) Owing to the narrowing of the liver-cells the spaces between them are much increased in size, and within these spaces the capillaries are usually found detached from these cells. It often occurs that only traces of the capillary walls remain. Blood is conspicuously absent in the capillaries in the immediate vicinity of the abscesses, but,

in its stead, these vessels are often partially filled with the same kind of granular detritus that surrounds them. Occasionally, in the neighborhood of the abscesses, small areas are found in which the liver-tissue is almost entirely replaced by blood. This is evidently the result of hemorrhage from some of the larger vessels. The walls of the intralobular veins and those of such other vessels as are not surrounded by masses of fibrous tissue become infiltrated with cells as the necrotic process advances toward them, and later swell, soften, and break down. Not infrequently the bloodvessels were surrounded by a slight, small round-cell infiltration, but there is a remarkable absence of anything like a general inflamma-

FIG. 5.

Degenerating liver-cells and an amœba. Toluidin blue and eosin. Beck ⅛, Oc. 3. Showing the various forms assumed by the degenerating liver-cells, and for the purpose of comparison an amœba is represented in the lowest figure. The liver-cells are stained red with blue nuclei, while the amœba is of an almost uniform dark-blue color.

tory reaction on the part of the tissues. In the tissues surrounding the abscesses, but never extending very deeply, numerous clumps of bacilli are seen, resembling in every way those found in the abscess-walls. The liver-cells, or the remnants of the same, which are in immediate contact with these bacilli are more completely broken down than the other cells in the vicinity, but this never extends to any distance.

It will be seen from the above, as pointed out by Councilman, that the soluble products of the amœbæ probably produce the extensive necrosis of the liver-tissue observed in the vicinity of the abscesses, but that complete breaking down and softening do not occur until the amœbæ come actually in contact with it.

The question as to how the amœbæ reach the liver has not as yet been

decided. Some have thought that they penetrated through the walls of the intestine, passed along the peritoneum, and burrowed their way through the capsule of the liver. This explanation seems to me extremely improbable. I think it all but certain that the amœbæ reached the liver by means of the portal circulation, as has been without doubt the case in instances recorded where there was a condition of almost miliary abscess of the liver. This view is also strengthened by the fact that the amœbæ are so frequently seen in the bloodvessels of the intestines.

Specimens of the kidneys were only examined in Cases XIV., XXII., and XXXV. In Cases XXII. and XXXV. the changes were limited to a slight granular degeneration of the lining-cells of the larger tubules, and there were very generally found collections of granular material, which stained faintly, in the spaces between the glomeruli and the capsule of Bowman. In Case XIV. the vessels were universally distended with blood. The cells of the tubules were intensely granular, their nuclei stained poorly or not at all, and they were frequently separated from their basement membrane and collected in masses in the centres of the lumina of the tubules. There was around the glomeruli none of the granular material described in connection with Cases XXII. and XXXV., but the nuclei of these bodies were considerably increased in number. There were no changes in the connective tissues of the kidneys, not even in the situations nearest to the liver-abscess.

There were no noticeable changes in the spleen.

After fixing specimens of the heart-muscle in Flemming's solution, many sections were made from Cases XIV. and XXII., for the purpose of determining as to whether fatty degeneration existed, but in neither was this found.

The lungs were normal in three of the four cases where autopsies were made. There were miliary tubercles in Case VI.

Symptoms. The disease usually begins acutely, with slight fever, griping, tenesmus, and frequent stools. Occasionally, however, the onset is gradual, there being a period varying from a few days to several weeks, during which the stools, at first only somewhat watery and frequent, become gradually more dysenteric in character, and finally the symptoms in the two varieties merge insensibly into each other. If the acute form becomes severe, we find, with the exception that here the inflammatory phenomena are generally more marked, the same symptoms that occur in the severe chronic form. The disease, however, as we commonly meet with it, is pre-eminently a chronic one, and lasts all the way from a few weeks to many years. In this variety the general condition is often remarkably good, but in the severer forms great emaciation is frequently observed. Loss of appetite is common, but nausea and vomiting rarely occur. Fever and rapid pulse are decided only in the severer forms of

the affection. The patients complain of a sense of uneasiness in the abdominal region, with a more or less constant desire to go to stool, as the disease is of a severe or mild type. After the affection becomes chronic, tenesmus is, as a rule, slight; but during exacerbations it is often quite severe. For the purpose of convenience we may—somewhat arbitrarily, it must be confessed—divide, clinically, the chronic form of the disease into several types.

1. *The very mild form.* The appetite and general health are good; no fever or acceleration of the pulse worth mentioning. The number of stools varies from two to five or six in twenty-four hours. This is the usual form in children.

2. *The moderately severe form.* The general nutrition is here decidedly interfered with, and there is frequently more or less anorexia. The pulse is somewhat increased in frequency, and there are irregular exacerbations of temperature, occurring especially at night. The stools vary in number from six or eight to fifteen or twenty in twenty-four hours.

3. *The very severe form.* This differs but little from the severest of the acute type. There are great emaciation, loss of appetite, and sometimes nausea and vomiting. Profuse sweating may occur. Hiccough is frequent. The extremities are often cold, and the pulse is rapid and weak. The temperature varies much, but is usually somewhat above normal; as death draws near it sometimes becomes subnormal. The stools are watery, filled with blood, mucus, and sometimes sloughs, and vary from twenty to fifty in twenty-four hours. It is really wonderful how long patients will remain in this condition without dying, and in no disease may death be more closely approached and recovery follow.

There are remarkable fluctuations in the severity of the symptoms in all of the chronic forms, and especially does this apply to the number of stools. In the milder types there are often not more than three or four actions in twenty-four hours, but at any time the number may be increased to ten or fifteen, or even more. On the other hand, in the severest forms the number of stools may without any apparent cause suddenly decrease in number, which may continue until complete recovery results. It thus happens that we often see the mildest forms of this affection suddenly changing to the worst, and *vice versa*. It is a fact that some cases are greatly aggravated by the taking of alcoholics —beer seeming to be especially injurious. Any form of bodily exertion is generally followed by an exacerbation of the symptoms. A constant aching pain in the back, not relieved by the recumbent posture, is a symptom which has been prominent in several of my cases, and has caused a great deal of trouble. Pain, of a tearing character, in the region of the colon, is also an occasional symptom. Œdema of the feet and face is occasionally seen.

In the milder forms the urine is, as a rule, normal, but albumin was

noted in some of the severer cases, especially if the temperature was high. In some of my worst cases I have day after day carefully examined the urine for peptones, by Hofmeister's method, but never found them present. Chlorides may be much diminished or suppressed. Their reappearance or increase is a most favorable symptom.

The feces in the milder forms may appear almost normal in both color and consistency; but, on careful inspection, small particles of bloody mucus will be found lying here and there on their surfaces. In the most severe forms the stools are loose, and contain considerable quantities of mucus and blood, mixed with partially digested food, are very offensive, and, while the color varies considerably, a more or less reddish tinge is quite constant. The discharges in the worst forms of this affection are, I think, entirely characteristic. They consist of a soft, jelly-like mass which has an almost evenly distributed pink hue. Sometimes the amount of mucus is not so great, and, especially shortly preceding death, they may lose almost entirely their mucous character and become scarcely more than bloody water. They are almost always of an alkaline reaction. In two cases I have found the reaction slightly acid. On microscopical examination, the feces are found to consist of fragments of partially digested food, much mucus and blood, pus, large epithelioid cells, multitudes of bacteria, and varying numbers of amœbæ. Cercomonas are not infrequently found.

The most important complication of amœbic dysentery is hepatic abscess. Out of seventy-eight cases it occurred fifteen times, one of which was in a boy. This complication occurs only in the early stages, or, at the most, it is certainly very rare in the chronic cases. It would seem that it is more common in some localities than in others, for in my thirty-five cases it has occurred only twice. This is, I am sure, in a measure explained by the fact that the majority of my cases were seen only after the disease had become chronic. The usual symptoms are chills, fevers of an irregular character, acceleration of the pulse, and a throbbing or aching pain in the region of the liver, or the pain may be reflected to the right shoulder. At the same time the tongue becomes coated, the appetite fails, and the patient rapidly loses flesh. On physical examination, there is found enlargement of the liver, and occasionally a fulness or marked protrusion develops at the lower border of the ribs. Jaundice is rare. Sometimes the usual symptoms are absent, and, with the exception of a general cachexia, no symptoms exist.

If the liver-abscess opens into the lung, as it very frequently does, abscess of this organ develops. The symptoms are cough and the expectoration of reddish-brown fluid, which, on microscopic examination, is found to contain amœbæ. The usual signs of consolidation develop in the lung. This complication I have not seen; it has occurred only three times in seventy-eight cases.

When perforation of the intestine occurs and peritonitis follows, the symptoms which are usually produced under these circumstances follow. I do not think, however, that the inflammatory phenomena are ever so marked as in the septic peritonitis which follows operations.

Appendicitis would, of course, occasion the usual symptoms. However, in my case, which is, so far as I know, the only recorded one of the kind, the symptoms were of such a mild character that the condition was not suspected before death.

Diagnosis. The diagnosis cannot be made with certainty except by finding the amœbæ. When we find, however, the patient suffering with a chronic dysentery that shows great variations in intensity, and which, moreover, resists all the usual remedies employed in the treatment of this affection, the probability becomes very strong that it is a case of amœbic dysentery, and should always lead to an immediate examination of the stools. Whether the amœbæ be the cause of this disease or not, I do not believe that it is possible to overestimate their value as a means of differentiating between this affection and conditions such as carcinoma and tuberculosis, for which it might be mistaken.

Prognosis. This is a very fatal disease. Out of the seventy-eight cases reported in this country there have been thirty deaths. It usually occurs in a milder form in people of the colored race than in those of the white. It seems very certain that the disease is rarely so severe in children as in adults. After it becomes chronic, and in the vast proportion of cases it does become so, the chances of recovery, even with the best treatment, are always bad. However, at any time the disease may rapidly grow better, and the patient recover, or, on the other hand, it may become worse, and death result. After apparent recovery relapses frequently occur, and a patient should never be considered out of danger until many months after all traces of the disease have vanished. Spontaneous recovery sometimes occurs.

Liver-abscess is a very fatal complication, twelve out of the fifteen cases reported in this country having proved fatal.

Abscess of the lung is very grave, but it probably adds little to the existing danger, as an opening is thus made for the pus pent up in the liver-abscess.

Perforation of the intestine and peritonitis are probably always fatal. As mine is the only recorded case of amœbic appendicitis, and as this case was complicated by a liver-abscess, nothing can as yet be said respecting the prognosis, but it is doubtless very bad.

Treatment. As I have never happened to see a case of this variety of dysentery at the very beginning, I can say nothing from personal experience as to the treatment. It would, perhaps, be best to employ the remedies which are usually used in the common catarrhal forms of this affection. Ipecac especially deserves a thorough trial, as it is

the drug that has proved of the most benefit in the dysenteries of India, which are probably often amœbic in character. In the chronic form I have from time to time used almost everything which has ever been recommended in the treatment of this class of diseases, but have rarely seen any striking effect produced by them. In its behavior toward drugs this variety of dysentery is almost as capricious and variable as are its symptoms. Opium checks the number of discharges, but I have never seen any permanent good result from it. Sulphuric acid has in several cases apparently produced some benefit, but never a cure. One patient recovered while taking salol. The treatment heretofore most recommended has been rectal injections of 1:5000 aqueous solutions of quinine sulphate. I used this treatment with great persistence in some of my earlier cases, but not in a single instance was there the slightest perceptible result. Injections of 100:300 watery solution of bisulphate of quinine were somewhat beneficial in one or two instances. One patient recovered while using injections of potassium permanganate, but in several other cases no effect was observed. In not a single instance did improvement follow injections of ice-water. Several years ago, while speaking on this subject with my friend, Dr. I. B. Diamond, at that time a student, he suggested that, on account of its antiseptic effect, possibly hydrogen dioxide might be of service. With but little hope, I must confess, I tried it on Case V., with the result that the patient rapidly improved. As soon as she was strong enough to go to work she did so, and at the same time abandoned the treatment and resumed a solid diet. In a short time she relapsed, and was again treated in the same manner with a like result. She again stopped the treatment, and, although I have not since seen her, I learn that she again has the trouble. Since that time I have treated many of my patients with this agent, with the result that some have been cured, and, with but one or two exceptions, the improvement after its use has been striking and immediate. All hemorrhage at once ceases, the stools rapidly decrease in number, and the general condition grows better. The effect of this drug, however, is by no means constant—it benefiting some patients much more than others. Even in the same patient this sometimes holds good, for, in one or two instances, in relapses, after it had apparently effected a cure, its use a second time was wholly without benefit. The ordinary commercial hydrogen dioxide is diluted from four to eight times with water, and the solution injected. About a quart is injected twice daily, which, after a week, may be gradually decreased. This is the only agent which has in my hands proved of any decided value. I do not doubt that it would prove of equal value in all varieties of dysentery, especially in the chronic forms.

Hydrogen dioxide, as found in the market, is notoriously uncertain in strength, some samples being utterly valueless. If improperly prepared

it decomposes rapidly. This may, in a measure, explain the lack of uniformity in the results obtained by its use.

The diet should be liquid, whether the affection is acute or chronic. I have used milk almost entirely, and, as a change, occasionally egg-albumin.

The remarkable toxic effect of the toluidin blue on the amœbæ would suggest the employment of rectal injections of this substance in the treatment of the disease; but in the absence of any knowledge as to its action on the human being, I have not made the attempt.

My friend, Dr. E. A. Corson, of Savannah, Ga., some years ago suggested to me that in suitable cases the establishment of an artificial anus at the ileo-cæcal valve, and the subsequent washing out of the colon with hydrogen dioxide, might be employed with advantage.

In finally dismissing the subject I cannot forbear stating my conviction that imperfect hygienic conditions are indirectly responsible for the prevalence of amœbic dysentery, and that with even an ordinary attention to sanitary laws the disease would in a short time cease to exist.

I wish, in conclusion, to express my thanks to my friends, Mr. J. C. Haskell, Jr., for the drawings of the fixed amœbæ, and to Dr. I. B. Diamond for much valuable assistance.

TWO CASES OF CONGENITAL SYPHILITIC CIRRHOSIS OF THE LIVER IN INFANTS.

By J. Michell Clarke, A.M., M.D. (Cantab.), F.R.C.P. (Lond.),
PHYSICIAN TO THE GENERAL HOSPITAL AND PROFESSOR OF PATHOLOGY, UNIVERSITY COLLEGE, BRISTOL, ENGLAND.

The following two cases, which came under my care within a short interval of each other, afford excellent examples of the changes in the liver in hereditary syphilis, in the one case more advanced than in the other, and leading to great destruction of the parenchyma of the organ:

The first patient was an infant of ten weeks; she was brought to the Bristol General Hospital with a history that she had appeared to be in pain in the stomach, that the belly had always been rather large, but that no distinct swelling had been observed until ten days previously, when a swelling in the upper part of the abdomen had appeared and had gradually increased.

During the same time the child had vomited all the milk she had taken. There had been no jaundice at any time, and no hemorrhage from the stomach or bowels; the child was fed at the breast; the bowels had been moved regularly, and the motions were normal. The urine was of normal appearance.

Both father and mother were healthy, according to the mother's statement, and had one other child, also healthy. The patient had suffered

from snuffles, but had had no eruption on the skin. The mother has since the patient's death given birth to an infant presenting the typical signs of hereditary syphilis. On examination the infant was very small, much emaciated, and appeared extremely weak and ill. The pulse was rapid, almost imperceptible, the fontanelle depressed. She was very anæmic, but not jaundiced. The temperature and respiration were normal, and the tongue clean. The abdomen in the epigastric and hypochondriac regions was the seat of a large swelling. Over this there was dulness and a large, hard, and resistant tumor was felt which moved freely with respiration, and appeared to be the liver; the lower edge of the tumor was sharp and smooth, and came about two inches below the umbilicus. The infant died on the following day.

At the post-mortem the body was extremely emaciated, there being no subcutaneous fat. The bases of the lung were congested, but otherwise the heart and lungs were normal. The abdominal cavity contained about three-quarters of a pint of serous fluid. The liver was very large and filled nearly the whole abdomen, extending from two to three inches below the umbilicus. It was finely granular on the surface, and of a pale yellow color.

On section it was smooth, pale yellow in color, but not bile-stained; of about the normal consistence and of a homogeneous appearance, the outlines of the lobules being indistinguishable. Scattered over the surface of the section were small, pale, rounded areas (miliary gummata).

The gall-bladder was empty and of healthy appearance; there was no obstruction of the bile-ducts.

The spleen was large, dark, and firm, and would weigh about three ounces.

The kidneys were pale; the capsules stripped well, leaving a smooth surface; on section, both cortex and medulla were very pale, but otherwise appeared healthy.

The intestines and stomach were contracted and empty, but otherwise normal.

Owing to the post-mortem being made at the patient's house, the weights of the organs were not obtained.

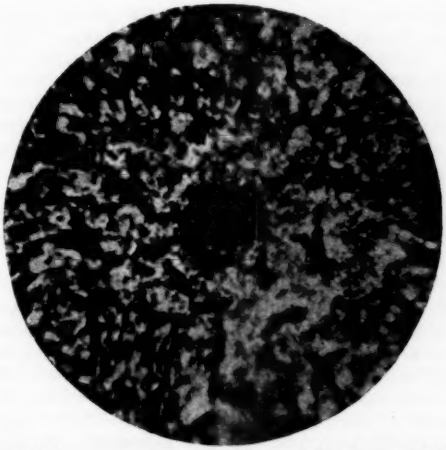
Microscopically, the small, pale areas in the liver are seen to consist of small collections of round cells (miliary gummata), seated, as a rule, in the periphery of the lobules. In some places the connective tissue in the sheath of the portal canals is much increased, so as to form a small mass of fibrous tissue. There is slight general increase of connective tissue in the portal canals around the vessels, and in many instances the newly-formed tissue is abundant around the central vein of the lobule. Both the vessels in the portal canals, the bile-ducts, and the branches of the hepatic veins appear healthy. With regard to the lobules the liver-cells show more or less imperfectly the arrangement into columns, but are flattened from the pressure of a new growth of richly nucleated, faintly fibrillated connective tissue along the walls of the capillary vessels, and often form Y-shaped figures, staining deeply; they are sharply demarcated from the surrounding newly-formed connective tissue, and their nuclei are oval or rounded and in great abundance. This growth pervades the lobules of all parts of the liver, but varies in amount in different parts. Where it is most abundant the liver-cells have almost disappeared, leaving conspicuous large round or oval nuclei, the product of cell proliferation, which is everywhere active, or the

cells are small, angular in shape, and atrophic. The protoplasm of the cells is finely granular, and more or less disintegrated, the outlines of

FIG. 1.

Under a low power. To show the miliary gummata, general appearance of sections and newly formed tissue within the lobules and in the portal canals, this figure should be viewed through a magnifying glass.

FIG. 2.



The same under a high power. There is a small gumma in the centre of the field, and the abundant formation of richly nucleated connective tissue between the hepatic cells, which are more or less atrophied, is shown. The sections were stained with carmine and methyl-aniline violet.

individual cells being unrecognizable. I could not trace the formation of connective tissue from liver-cells. (See Figs. 1 and 2.)

The process is thus one of an active new formation of a richly nucleated connective tissue along the intralobular capillaries, due to proliferation of the connective-tissue cells between the capillaries and the liver-cells and of the capillary endothelium, with consequent degeneration and atrophy of the proper cells of the organ.

The other patient was a male infant of one month. He had been extremely delicate from birth, and was taken ill one day before admission, with vomiting and cough. He had not had snuffles nor any skin eruption.

Soon after her marriage the mother had a sore-throat and a measly rash over the trunk and limbs, and since then had suffered much from headache and aching pains in the limbs. The child was very ill on admission, with a temperature of 101° F., rapid respiration, depressed fontanelle, and anæmic appearance. There was no jaundice.

At the base of the left lung there were dulness and bronchial breathing, and moist râles were audible all over the chest. The upper region of the abdomen was prominent, and the liver was felt to be enlarged. He died the day after admission. At the post-mortem the body was much emaciated and weighed eight pounds twelve ounces.

There were no scars or eruption on the skin.

The lungs showed scattered patches of broncho-pneumonia, with general bronchitis. The heart was healthy; it weighed two ounces. The abdomen was large; there was no ascites, and the peritoneum was normal. The stomach and intestines were pale, but healthy. The liver weighed seventeen and a half ounces. It was smooth and pale yellow in color, and its edge sharp. On section it was smooth, pale yellow, but in parts a yellowish white; it was very tough, not breaking down under the finger. In the central parts were small tracts in which the proper substance of the liver was replaced by smooth fibrous tissue in small bands and masses. Around these areas of fibrous tissue the lobules were recognizable, and the substance of the liver was softer and yellowish in color. The gall-bladder was empty, the bile-ducts normal.

The spleen, weighing two ounces, was smooth, somewhat soft, dark red on section, mottled over with small areas of extravasation. The suprarenal capsules were healthy.

The kidneys were pale; on section the boundary zone was injected, and the cortex and pyramids were pale—otherwise they were normal. The brain was normal, but very pale.

On microscopical examination the proper structure of the liver was much altered, but not uniformly so, by a diffused change, consisting of a formation along the capillary vessels of a lowly organized, nucleated connective tissue, spreading through the lobules from periphery to centre, with nuclear proliferation of the cells along the capillary walls, but without marked round-celled infiltration. The liver-cells were throughout the organ greatly degenerated, and atrophied from pressure. Their nuclei were proliferating, and the cell substance was filled with fine granules; in a few places they had undergone fatty degeneration, but this was not a marked or general change. These appearances were less marked in some parts than in others; in some the liver being hardly

recognizable, in others being present but highly granular. The arteries showed endarteritis obliterans, and the epithelial cells in the bile-ducts were also proliferated. The veins were normal.

Along the portal canals and between the lobules, also in some parts of the liver around the central vein of the lobule, there was an increased amount of connective tissue, staining badly and of a pale, translucent, homogeneous appearance. This was apparently newly and imperfectly formed connective tissue. In a few places there were very small collections of round cells (interlobular), but these were few and inconspicuous.

In the kidneys the cortical epithelium was granular, and the small and medium-sized arteries showed endarteritis obliterans. This did not affect the arterioles or the afferent vessels of the Malpighian capsules.

This case, then, showed the same changes as the first, but in a more advanced stage, with much more extensive destruction of the liver-cells; differences were found in the absence of miliary gummata, unless the few small collections of round cells mentioned above may be taken as remnants of them, and in the presence of endarteritis obliterans. This latter process might be answerable in part for the greater destruction of the hepatic cells, although the chief factor must be held, as in the first case, to be the pressure exercised upon them by the newly-formed tissue along the intralobular capillaries.

Thus the process is one of a subacute interstitial hepatitis resulting in the progressive destruction of the proper cells of the organ. If the term cirrhosis be applied to the condition it should be described as an intercellular or intralobular cirrhosis. It differs from the acute form of biliary cirrhosis occasionally met with in infants,[1] in that the process in the latter is a much more acute one, starts from the small bile-ducts, and is attended with jaundice. Clinically, the points in the above cases were the history of syphilis, the absence of jaundice, the large size of the liver, the extreme degree of emaciation, and the vomiting of all food. In the second case death was due to the pulmonary complication, but in the first there was no complication, and the early age at which death occurred and the acute character of the symptoms of the terminating illness are noteworthy.

For the photographs I am indebted to the kindness of Mr. James Taylor.

[1] See a paper by Gibbons, of Calcutta: "Scientific Memoirs by Medical Officers of the Army of India:" 1891, vi., and by author, Brit. Med. Journ., June 30, 1894.

GLAUCOMA AND THE INFLUENCE OF MYDRIATICS AND MYOTICS UPON THE GLAUCOMATOUS EYE.

By Edward Jackson, A.M., M.D.,
PROFESSOR OF DISEASES OF THE EYE IN THE PHILADELPHIA POLYCLINIC; SURGEON TO WILLS' EYE HOSPITAL, PHILADELPHIA.

Mydriatics in Glaucoma.

It is well known that the application to the eye of one of the mydriatics has sometimes been followed by an outbreak of glaucoma. The following case is the only one of the kind which I have seen among several thousand patients whose eyes were subjected to the use of different mydriatrics for the determination of errors of refraction.

Case I.—A dark mulatto woman, aged twenty-five years, applied at the Philadelphia Polyclinic at the end of the clinic hour, giving this history: Two years before the left eye became red and painful. Since then she had pain in it at intervals, and blurred vision. The eye weeps continually. She was illiterate, and her vision was not obtained at the first visit, nor was any record made of an ophthalmoscopic examination.

A solution of duboisine sulphate (1 to 240) was ordered to be instilled in both eyes to prepare for measurement of her refraction.

Next day four instillations had been made. Her pupils were well dilated, and accommodation paralyzed, but there was marked arterial pulsation in each eye. In the right eye there was a wide physiological cup with sloping sides. In the left eye the cup was as wide as the disk, with abrupt edges, and about 2.5 dioptres deep. The central artery bifurcated in sight; and the principal branches showed an arterial pulsation extending beyond the disk margin. Tension + T. doubtful in each eye. She had no pain since using the mydriatic. There was no hyperæmia of the globe.

There was no failure of the arterial pulse on raising the arm. She had muscular rheumatism two years previously, but not affecting the joints, and was not seen for it by a physician. She was carefully examined in Dr. Cohen's clinic, but no evidence of cardiac or vascular disease was detected. Vision was: Right, + 1.5 sph. = $\frac{1}{20}$ mostly; left, with + 1.25 sph. = $\frac{1}{70}$.

The use of the duboisine was immediately stopped, but two days later the eyes began to give pain; and when she came on the fourth day there was haziness of the dioptric media, and a red pericorneal zone, the left eye being the worse.

Sixth day. Right eye free from pain; media clear; but the arterial pulsation still marked. The left eye showed a marked pericorneal zone; fundus dimly seen; tension of each eye was now + 1. She was put under the use of eserine solution (1 to 240), instilled three times a day.

Eighth day. The pupils are still fully dilated. The right eye still shows slight arterial pulse. The left eye is free from pain, with slight pericorneal zone, the media clearer, uncertain pulsation. Tension: Right, + T.; doubtful. Left, + T.; doubtful.

Tenth day they continued the same.

Eleventh day. Left pupil beginning to contract; pulsation now clearly seen in that eye.

Twenty-first day. Right eye, tension +; media hazy; marked arterial pulsation; pupil 6.5 mm. in diameter. The left eye is as before, with pupil 4.5 mm. in diameter. She is still using eserine.

One month after use of the duboisine vision in the right eye = $\frac{15}{30}$, and in the left eye = $\frac{15}{100}$.

She persistently refused to allow an iridectomy, and several months afterward had become practically blind in the left eye, with the right growing worse with repeated glaucomatous attacks.

The first thing to be noted with regard to this case is that careful ophthalmoscopic examination, with less reliance on the age of the patient and her race, might have led to the recognition of the glaucoma at the first visit. Glaucoma at so early an age is very rare. I have seen no other case in my own practice, and can recall but one seen in the service of a colleague. Priestley Smith[1] found among 1000 cases of primary glaucoma but five patients under twenty years of age, and but twenty-three between twenty and thirty, and it is probable that of cases occurring at such an early age a much larger proportion are reported than of those occurring at the usual time after middle life.

Again, glaucoma is rare among colored people at any age, so that it was natural not to have thought of glaucoma in connection with the case. It is probable, too, that a hasty ophthalmoscopic examination was made, and nothing abnormal detected. Yet it is almost certain that the cupping of the left disk, recognized the next day, might have been made out by careful study through the undilated pupil; and the history, although fitting well with the supposition of eye-strain, when read by the light of subsequent events, pretty clearly indicates that the left eye had for two years been the seat of occasional mild glaucomatous attacks.

The case is of especial interest, because seen from the very beginning of the attack. On the first day after the use of the mydriatic the increase of tension was doubtful, and there was no positive sign of glaucoma, except the pulsation of the retinal arteries, and the cupping of the disk in the left eye. Not until two days later was there any pain, redness, or marked increase in the intraocular tension.

The length of this interval between the use of the mydriatic and the occurrence of marked symptoms of glaucoma was exceptional, the attack so produced usually becoming severe within a few hours. Still this delay has been noted in other cases. F. T. Rogers[2] reports a case of acute glaucoma in both eyes following the instillation of homatropine, in which it was not until the fourth day (when the influence of the homatropine has usually entirely passed off) that marked increase of the tension was

[1] Trans. Ophthal. Soc. of United Kingdom, vol. vi. p. 294.

[2] Ophthalmic Record, v. p. 421.

found. In a case reported by S. Snell,[1] the eyes were examined the day after that on which atropine had been used, and were found "perfectly normal." But three days afterward the sight was affected, and a sharp attack of glaucoma occurred.

The case also seems to illustrate the value of arterial pulsation as the earliest and most delicate test of increased tension in the eyeball, at least in some cases. When pulsation was discovered the absence of decided increase of intraocular tension or other characteristic symptoms of glaucoma gave the impression that the pulsation was due to diminished blood-pressure in the arteries. A careful search was made for evidence of probable causes of diminished arterial tension, and only when these were proved to be entirely absent was it thought probable that the pulsation was due to increased tension in the eyeball.

G. C. Harlan has reported a case[2] in which transient pulsation of the retinal arteries was induced by the application of homatropine, but in which no other symptom of glaucoma developed. The patient, a man, aged sixty-six years, with tension regarded as normal or of doubtful increase, showed after the application of homatropine a vigorous pulsation in the retinal arteries of one eye, which pulsation ceased when the pupil was contracted again with eserine.

After this observation had been made there was noticed some enlargement of the anterior ciliary vessels, but no other symptom of glaucoma. The instillation of homatropine was repeated three times, each time producing decided pulsation, which promptly disappeared when the pupil was contracted with eserine.

It must not be supposed that all cases of glaucoma are certain to be injuriously affected by mydriatics. The writer has seen several cases in which mydriatics have been used without causing increased pain. In one case, seen in 1883, which had probably been treated for several weeks with atropine, the patient after iridectomy regained vision, which at the end of four years equalled fifteen-fifteenths partly, although the field was greatly restricted. In this case, at the end of nine years vision began to fail, and, in spite of the use of eserine, was reduced to mere light perception, when she was last seen in 1894.

O. F. Wadsworth[3] reports a case of hemorrhagic glaucoma in which the pain diminished for a few days under the use of atropine, but subsequently returned, and the eye was enucleated. He also reports the following:

"A woman came to the hospital complaining of pain in and about one eye. There was circumcorneal congestion and one or two narrow posterior synechiæ. Atropine was applied. Half an hour later the

[1] Trans. Ophthal. Soc. of United Kingdom, vol. ii. p. 100.
[2] Transactions of the American Ophthalmological Society, 1890, p. 653.
[3] Ibid., 1880, p. 150.

pain was relieved, and an ophthalmoscopic examination revealed strong arterial and venous pulsation and a large central excavation. Tension was increased. On inquiry into the history it was learned there had been short glaucomatous attacks during the past five months, of late at intervals of less than two weeks. In accordance with the prevailing opinion, the patient was warned against the use of atropine; but on a subsequent attack, ten days later, she did use atropine herself, and again with relief of pain. An iridectomy stopped the attacks."

G. C. Harlan has known duboisine, used by mistake for eserine, to give relief, and W. F. Norris says it is well known that atropine does not always increase tension.[1]

The reason for such an apparently contradictory effect produced by mydriatics in glaucomatous eyes is probably that pointed out by Priestley Smith,[2] "That whenever a mydriatic raises or lowers the tension of the eye in any very marked degree it does so by altering the position of the iris in such a manner as to hinder or to promote the escape of the intraocular fluid."

One effect of dilatation of the pupil is some thickening of the iris opposite the filtration angle of the anterior chamber, and when this angle is already narrowed or partly obstructed, such thickening increases the obstruction. But while mydriatics show a liability to increase the obstruction of the filtration angle, and thus to raise the tension of eyes already glaucomatous or upon the verge of glaucoma, they probably have no more general tendency to cause glaucoma. And they may, by reducing intraocular congestion or in other ways, exert a beneficial influence upon glaucomatous eyes that they cannot injure by dilating the pupil.

The liability to do harm in glaucoma appears to be shared by all the mydriatics. As each new drug of this class has been introduced the hope has been expressed, and sometimes apparently sustained by the history of the earlier trials, that it would not have this influence in glaucoma. But subsequently cases of this kind of harm from its use have been reported.

In practice, however, the two mydriatics, homatropine and cocaine, may be regarded as free from serious danger of causing permanent injury to the patient, since their effect upon the iris can be promptly overcome by eserine. Thus, in Dr. Harlan's case, referred to above, the arterial pulsation was entirely under control through eserine, and during the last few years the use of cocaine (with one of the myotics to prevent any dilatation of the pupil by it) has grown rapidly in favor as a means of treating glaucoma where, for any reason, iridectomy cannot be at once resorted to.

[1] Ophthal. Record, 1897, p. 319.
[2] Ophthalmic Review, 1882, p. 79.

As I have found, experimentally, in a limited number of eyes,[1] eserine is capable of neutralizing the effects of six times its weight of homatropine upon the iris and ciliary muscle, so that by repeated instillations of a strong solution of eserine (1 to 240) it is possible to contract the pupil within a few hours after it has been fully under the influence of homatropine. This is in strong contrast to the influence of atropine, which overcomes five, or duboisine, which overcomes twelve, times its weight of eserine.

The application of the eserine, however, should be made as soon as the glaucoma is discovered. The action of homatropine is commonly limited to two days in normal eyes, but may be prolonged to five days (see the case reported by de Schweinitz);[2] and is specially liable to be prolonged in glaucomatous eyes, as in the case reported by Rogers. Then, too, the increase of tension interferes with the absorption of the drug, and the longer the pupil is allowed to remain dilated the more the intraocular pressure is likely to rise.

With cocaine the case is somewhat different. In the first place, it requires but one-twenty-fifth its weight of eserine to neutralize its action on the pupil. Even one-fourth of its weight of pilocarpine is sufficient to do the same thing. And aside from its action upon the iris, dilating the pupil, the tendency of cocaine is to diminish the intraocular pressure by causing constriction of the bloodvessels. In a combination preventing its dilator effect on the pupil it is a perfectly safe drug to apply to the glaucomatous eye.

The Influence of Myotics in Glaucoma.

In 1876 Laquer published his observations on the curative power of eserine in glaucoma.[3] In the same journal for August 12th, of that year, a communication from Dr. Lucius claimed that A. Weber had employed eserine for this purpose in 1873 and 1874, and that the case treated with it in the latter year had remained permanently cured. However, Weber's paper,[4] published next year, cautions against certain dangers from eserine, and urges pilocarpine as a better and safer remedy for glaucoma.

Since that time these myotics, especially eserine, have been widely tried, and their influence in contracting the pupil and diminishing the intraocular tension in glaucoma has been generally recognized. But it has also been recognized that the cases they will permanently cure are very few, and that there is danger in the reliance that may readily be

[1] Chairman's Address before the Section on Ophthalmology of the American Medical Association. See Transactions of the Section, 1895, p. 21.

[2] Ophthalmic Review, 1892, p. 1.

[3] Centralblatt für med. Wissensch., June 10, 1876.

[4] Graefe's Archiv, 1877, xxiii. p. 1.

placed upon them on account of the marked benefit they afford in the earlier stages of the disease. This danger is illustrated by the following case, reported in this JOURNAL for April, 1882, by the late A. G. Heyl, who saw it in consultation with F. P. Henry, who years later referred the same patient to me.

CASE II.—Mrs. M., aged fifty-five years, had duboisine sulphate instilled into her left eye by Dr. Henry, to facilitate the ophthalmoscopic examination, the vision in that eye being 10/L, and the tension of the eyeball normal. With the ophthalmoscope a suspicious cupping of the disk was discovered. Within twenty-four hours acute inflammatory glaucoma developed, the eye became injected, the cornea and vitreous hazy, so that no view of the fundus could be obtained, and the tension considerably increased. Within forty-eight hours after the use of the duboisine Dr. Henry made a large upward iridectomy, and the glaucoma was perfectly cured. The other eye appeared normal, with a physiological cupping of the disk; but about two weeks later it became hard, with diminished vision. Under the use of eserine and morphine these glaucomatous symptoms promptly subsided. A month after the operation the patient had vision of twenty-fiftieths in each eye, and probably would have shown better vision with carefully chosen glasses. Both eyes had become entirely quiet.

In June, 1896, fifteen years after the original trouble, the patient, who had not for some time been under his care, again sought advice of Dr. Henry, and was referrred to me.

August 1, 1896. For some years after the first attacks of glaucoma her eyes had remained well. Then the right eye began to suffer from rainbow rings around the light, and obscuration of vision. She then resorted to eserine, which promptly relieved the attack. But such attacks recurred and gradually grew worse. She still used the eserine, which partly preserved its beneficial influence. But the eye continued to grow worse with each attack, until more than a year ago all vision had been lost.

She now presented in the right eye a dark pericorneal zone of redness and greatly injected anterior veins. The anterior chamber was almost obliterated. The pupil was 3 mm. in diameter, and fixed. The media were hazy, leaving a fair fundus reflex, without any details of the fundus being visible. The right eye is constantly and extremely painful, the pain impairing her general health. She came for relief from pain, which eserine no longer helped, although it continued beneficial up to a few months ago, and she still continued to use it. The tension of the eyeball was + T. 2.

The left eye is free from redness and presents an upward iridectomy of $\frac{1}{6}$ of the iris. There is incarceration of the iris at the angles of the wound, and at the outer angle a slight pigment mass, small cystoid cicatrix. The anterior chamber was very shallow, the lens slightly hazy.

August 3*d*. With the hope of saving the eyeball, the patient was etherized and a large upward iridectomy done on the right eye. One-fourth of the iris was removed, and the lens was extracted. One hour later great pain in the eyeball set in, and subsequently there was evidence of bleeding. The next day there was a large clot in the conjunctival sac, and the vitreous and shreds of retina were hanging from the wound.

The vitreous and the retina with clot were cut off and the lips of the wound drawn together. The globe, however, continued hyperæmic. The upper margin of the cornea became somewhat infiltrated, and the infiltration extended slowly downward. The eye continued very painful.

20*th*. The patient was again given ether and the eyeball removed. Healing was subsequently normal; the pain relieved.

The left eye continued well. Careful ophthalmoscopic examination showed slight striation in the periphery of the lens, the choroid patchy and thinned, the optic disk of good color, with a cup extending almost its whole width, 1 to 1.5 D. deep, with abrupt edge upward and inward, but not downward and outward. The retinal vessels appeared normal.

The ophthalmometer showed 2.5 D. of corneal astigmatism; but by skiascopy and with test lenses her correcting lens was found to be —2.5 D. sph. ○ + 6 D. cyl. ax. 145°, with which vision = $\frac{4}{6}$ full.

A careful study of the field of vision with tests 1 centimetre square showed no perceptible narrowing of the field in any direction for either form or color.

This patient, with both eyes strongly predisposed to glaucoma, had a mydriatic used in what was then decidedly the worse eye of the two, producing a rather violent glaucomatous outbreak. But this being recognized promptly and treated efficiently, she has, as the final outcome of the disease in it, a good and serviceable eye sixteen years afterward. The other eye, showing some signs of the same condition, was treated with eserine, with the most excellent immediate result. Indeed, months afterward the case was accurately reported as one of a cure of one eye by eserine, equal in all respects or superior to the cure of the other by iridectomy. Nevertheless, the increased tension returned, and, running the usual course with exacerbations, each of which at first yielded promptly to the eserine, the result was in the end complete blindness and the necessary enucleation of the eye for the relief of intolerable pain.

The following case also illustrates the dangers of over-confidence in the efficiency of eserine, and of the yielding to the natural inclination to avoid an operation:

Case III.—Miss T., aged about fifty-five years, suffered during February, 1882, from a sharp attack of facial erysipelas, with great swelling of the eyelids and suppuration of the lids of the right eye. In the latter part of the month, while convalescing from this attack, she was suddenly affected with acute inflammatory glaucoma of the left eye.

I saw her in consultation with Dr. Jacob Price, of West Chester, on the 28th of the month. In the left eye vision was reduced to counting fingers at eighteen inches; the pupil was widely dilated; the anterior chamber shallow, the cornea, and also the lens, hazy. The right eye had vision of four-twentieths, with cortical opacities in the periphery of the crystalline lens.

Iridectomy was advised and urged, but was not consented to and performed until March 14th, when the upper fifth of the iris was removed up to the ciliary border. Healing was prompt and normal. No relapse of the erysipelas occurred.

One week after the operation on the left eye, the right eye showed impaired vision, dilated pupil, and increased tension. Under eserine it promptly returned to normal tension, and vision of four-twentieths. After this the eyes continued normal, and vision remained unchanged for eighteen months, although there was increasing opacity in the periphery of each lens. Vision in the left eye had only risen two two-hundredths, with a very small visual field.

The patient refused to have her good eye operated upon, passed from under my care, sought other advice, and was given the opinion by a prominent ophthalmic surgeon that she was suffering from incipient cataract, and that neither eye presented the slightest symptom of glaucoma, which opinion she showed in writing for my edification.

In October, 1888, I was again asked to see her, and found each eye presenting an almost mature cataract. But the right eye was absolutely blind, and of stony hardness. The left eye had good quantitative perception of light, and no decided increase of tension. Her history of the interval showed that reliance had been placed on myotics, and these had for a time given relief. But the disease had slowly run its course in the right eye to complete blindness, with very great pain. The pain had now, however, subsided.

At the patient's solicitation the cataract was extracted from the left eye, giving improvement of light perception, but no useful vision.

In this case, with one eye already practically lost and the other free from any manifestation of glaucoma for many months after the use of a myotic, there was great reason to hesitate about urging iridectomy. Yet the preservation for several years of some vision in the eye upon which iridectomy had been done, after it had been so badly damaged to start with, seems to indicate distinctly that an early iridectomy might have saved useful vision in the right eye throughout life.

The physician, accustomed to trust to the influence of drugs and the recuperative powers of nature, and knowing that myotics do exert a markedly beneficial influence in many cases of glaucoma, is very likely to feel that the urgency of the ophthalmic surgeon, to open the eye and cut away a piece of the iris in such cases, is evidence of an excessive desire to operate. In this instance nothing could be further from the truth. The well-informed ophthalmic surgeon, knowing the insidious and deadly character of the affection, would usually be very willing to see his glaucoma patients go to some one else for either advice or operation. The fact is, he knows the persistent, progressive malignancy of the condition; and if he fails in the performance of his whole duty it is quite as apt to be by refraining from urging strongly enough an operation that he knows might also possibly fail to give permanent relief, and so might bring discredit upon him.

It is quite noticeable that writers on ophthalmology, in the last few years, while recognizing the influence of myotics in the palliation of glaucoma, almost without exception warn their readers against putting

their trust in these remedies as a means of curing or of permanently holding in check the morbid process.

The number of cases of glaucoma reported as cured by eserine and pilocarpine is not very large, and most of these have been reported within one or two years after the cessation of the glaucomatous attacks. It will be noticed that either Case II. or Case III., as given above, might have been equally well reported as a cure with eserine two years after the cure was supposed to be complete.

S. Snell reported a case of a glaucomatous attack caused by atropine where the eye remained well seven years afterward; but the patient, going to India, lost his sight within two years, although it was only probable that this was due to glaucoma. I have operated for an acute glaucomatous outbreak upon an eye still retaining useful vision ten years after a positive diagnosis of glaucoma had been made, no treatment having been resorted to during that interval. And Priestley Smith reported a case[1] in which the positive diagnosis of glaucoma was made by William Bowman nearly twenty years before operative treatment was undertaken, yet iridectomy gave an excellent result in each eye. Evidently, two or three years is not long enough time to establish the permanence of a cure of glaucoma. On the other hand, there have been a few cases reported of apparently permanent cure. Thus, Pye Smith narrates one[2] of a single outbreak following the use of atropine, where the patient remained free from symptoms of glaucoma until her death, about six years later.

It is noticeable that in all cases of apparently permanent cure of glaucoma by myotics the drug has been resorted to at a very early stage of the disease. In a large number of cases it gives, at this early stage, prompt relief from the attack. Later, its influence is still markedly beneficial, but the relief is not complete; and at a still later stage it loses all power of diminishing intraocular tension or lessening the severity of any of the symptoms of glaucoma.

The earlier writers upon the subject found eserine, like other remedial measures, less efficient in simple than in inflammatory glaucoma, and many regarded it as useless in simple glaucoma. Later observations seem to show that in the earlier stages of simple glaucoma it may beneficially influence the course of the disease. Thus, de Schweinitz[3] reports a case in which, under its use, there was a gradual widening of the fields of vision and complete restoration of the field previously lost.

Zentmayer and Posey[4] conclude, from a study of 167 cases, "that the effect of the administration of eserine and of the performance of iri-

[1] Ophthalmic Review, 1885, p. 261.
[2] Trans. Oph. Soc. of United Kingdom, vols. ii. and vii.
[3] Ophthalmic Review, 1894, p. 222.
[4] Wills Eye Hospital Reports, 1895, p. 82.

dectomy in checking the course of the disease is proportionately the same in the treatment of simple glaucoma. As operative procedures are always to be deprecated when other measures are equally valuable, eserine should be employed in all cases of the disease." But their cases were not under observation long enough to test the permanence of the benefit.

SUMMARY. With reference to the use of mydriatics, we are justified in saying:

In general, they should not be applied to eyes that are glaucomatous or upon the verge of glaucoma. In such eyes the dilatation of the pupil they commonly produce is dangerous, and may cause increase of intraocular tension, which, if not speedily relieved, will do permanent damage. But the risk of this effect from a mydriatic is not to be guarded against by fixing an age limit before which mydriatics may be considered safe and after which they should not commonly be employed. In the great majority of eyes a mydriatic cannot cause glaucoma at any time of life, while, on the other hand, a few patients are affected with the disease even from childhood. The danger is best guarded against by bearing in mind the symptoms of glaucoma, and always looking for them before ordering a mydriatic, especially by a careful ophthalmoscopic examination.

In very rare cases careful examination may not reveal the imminence of glaucoma, yet when the mydriatic has been used the outbreak may occur. In such a case the usual remedies for glaucoma should be promptly resorted to. The mydriatic should be stopped and iridectomy strongly urged. With the proper iridectomy promptly done the prognosis for complete permanent cure is excellent, the results being decidedly better than in cases discovered at a later stage when the glaucomatous outbreak has occurred spontaneously. Indeed, if the patient permits the proper, immediate treatment of his case, the fact that an outbreak of glaucoma has been evoked by the use of a mydriatic is probably a cause for congratulation rather than for regret. For the eye was, in all probability, doomed to the disease, and the earlier application of the remedy gives the better chance for complete and permanent cure.

I believe it would be perfectly proper, after explaining the matter to the patient, and getting his assent to prompt iridectomy if it should be indicated, to use homatropine as a test for the presence of glaucoma at the earliest stage in doubtful cases.

If the patient refuses iridectomy, eserine should be promptly resorted to in such strength and with such frequency as may be necessary for the reduction of the pupil. In the case of a glaucomatous outbreak following the use of a mydriatic more persistent in its action than homatropine, it would be proper to shorten the period of mydriasis by tapping

the cornea and evacuating the aqueous humor, preparatory to the efficient use of eserine.

In any case of glaucoma in which the pupil is firmly bound down by adhesions, or is otherwise so fixed that mydriasis cannot cause thickening of the iris opposite Fontana's space at the angle of the anterior chamber, especially if the application of eserine aggravates the symptoms, it is justifiable to apply atropin or some other mydriatic, and in a small proportion of cases such applications will be of marked benefit.

Myotics are beneficial in glaucoma only when the pupil is still movable; that is, chiefly in the earlier stages. When not beneficial, they are usually distinctly injurious.

If for any reason iridectomy cannot be done, myotics are always to be tried in the earlier stages of the disease. If they cause marked improvement they may be continued so long as they cause improvement. If they reduce the eye tension they may be continued so long as they keep the tension down; if they promptly relieve attacks they may be continued so long as the attacks are rendered less severe and frequent, and leave no permanent impairment of function, either of central vision or of the field, in the interval. But in the vast majority of cases there will come a time when the influence of the myotic, although still favorable, is less favorable than it has been; and after this it is liable rapidly to lose its power to do any good at all. Hence, whenever this period arrives the patient should be warned that the myotic is insufficient, practically worthless, and an operation, preferably iridectomy, gives the only chance for escaping complete blindness, and, perhaps, intense suffering.

THE DESCRIPTIVE ANATOMY OF THE HUMAN HEART.

By William Keiller, F.R.C.S. Ed.,
PROFESSOR OF ANATOMY, UNIVERSITY OF TEXAS.

The magnificent work of Braune and His has, within the past decade, revolutionized many sections of descriptive and topographical human anatomy. Till lately, dissection of the fresh or imperfectly-prepared subject was the only means used to arrive at a knowledge of the form and relations of each structure; but within recent years the examination of the body made while frozen, and the dissection of subjects hardened to comparative rigidity by continued intra-arterial infusion of such hardening agents as chromic acid, chloride of zinc, or corrosive sublimate, have corrected grave errors which had arisen from the exclusive study of the organs in the flaccid condition met with when ordinary methods are used. Even on the post-mortem table, and

within thirty-six hours of death, the liver, spleen, and heart "flop" out of all shape (I can think of no more expressive word, if it be somewhat inelegant), and are no more like the same organs as seen when hardened *in situ*, than the jelly-fish, half embedded in the sand—a shapeless and repulsive mass—resembles the living embodiment of symmetry and beauty propelling itself through the clear blue water. What, then, must be the condition of the same organs weeks or months after death, in bodies preserved by freezing or by arsenical solutions which do not harden the tissues? Verily, such organs are the despair of the medical student, and those who have never seen hardened organs may well be excused should they regard the models of His and allied descriptions with a certain amount of respectful incredulity. Such, I confess, was my own feeling toward them till, on the adoption of my present method of preserving bodies, I found to my surprise that these and other organs were thereby sufficiently hardened to present almost perfect counterparts of these models, without any interference with the methods of dissection. Thus there is now in my dissecting-room scarcely a liver, kidney, or spleen removed by the student from his subject that does not exhibit in perfection all the surfaces, borders, and impressions described and modelled by His.

While the text-book descriptions of liver, kidney, and spleen have been changed to accord with recent views, it is somewhat strange that the heart is still portrayed as it used to be many years ago; and that, though it requires the most vivid imagination and elastic conscience to reconcile the description with the accepted model of His; and this is still more strange, since clinicians have found the text-book descriptions of the organ so utterly inadequate that they have had to invent terms of their own in order to indicate anatomical facts in the living—terms which have no place in the dissecting-room vocabulary. I have long felt that, though the description of the heart as presenting a base, apex, right and left borders, and anterior and posterior surfaces is sufficient to describe the flabby, shapeless mass one finds on the post-mortem table when the excised organ is there before one, yet it is useless as applied to His's model or my own specimens hardened *in situ*. I have, therefore, for the past few years described in my lectures what I saw before me, without reference to the text-book; and I feel that the matter is of sufficient importance to merit presentation to my brother anatomists. When I consider that my own description is immensely more complex than that now in vogue, I feel some little compunction in suggesting an additional burden to the already sorely-laden student of anatomy; but I am encouraged by the remembrance of what has happened in the case of the liver. That organ, which, in the primitive simplicity of old text-book descriptions, had only two borders, two surfaces, and no impressions at all, has now five borders, as many surfaces,

and eight impressions; but everything is so definite, its relations are so evident, and the picture can be so vividly printed on the brain that what was before a pure matter of memory and a puzzle to the student, is now easily described and readily understood and remembered.

First, let me allude to the inconsistencies in our accepted descriptions. The pericardium is spoken of as cone-shaped, its apex upward, its base resting on the diaphragm. Yet the heart, which, with the commencement of the great vessels, completely fills it, is described as a cone with its apex directed downward, forward, and to the left, and its base upward, backward, and to the right. But this is a small matter, and probably justifiable; graver inconsistencies are to come.

Clinical manuals, such as Gibson and Russell's *Physical Diagnosis*, and articles on topographical anatomy, such as that in Morris's *Treatise*, discard the word "base," or explain that in their use of it they mean the "upper limit;" use "lower border" to signify what in the dissecting-room we call the "left border," apply the term "right border" to what in the dissecting-room we do not describe at all, and their "left border" is anything but synonymous with the left border of the descriptive anatomist.

Having, I trust, succeeded in showing that some change is necessary, I shall endeavor to submit to you a description of the external configuration and relations of the human heart which shall be accurate, concise, and free from redundancy. I would beg that those who honor my description when in print with a careful perusal, will compare it and the illustrations with the model by His, and those who desire to verify it in the dissecting-room may do so in any body injected through the right common carotid or femoral artery with a gelatine injecting mass.[1] The drawings are made from a rather enlarged specimen, of which the right side was filled with coagulated blood and the left side with injection mass; but, though large, it agrees in all essential matters with His's model and many other dissecting-room specimens I have carefully examined. My description is markedly borne out by vertical sagittal mesial and vertical coronal sections of the frozen body; horizontal section I have not yet been able to compare it with.

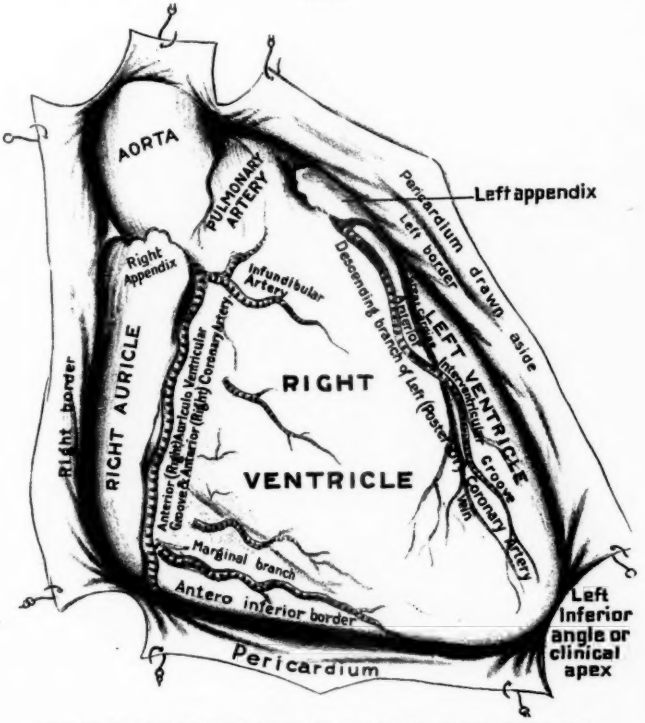
Thus viewed, the heart is an irregular, four-sided pyramid, whose base rests on the diaphragm, and whose apex has been, as it were, removed to afford attachment for the ascending trunks of the great vessels. It thus presents for examination five surfaces (including the base), borders separating these, an anatomical apex, and a "clinical apex," a term which I feel compelled to retain because it is almost inseparable from physiological and clinical phraseology.

The anterior surface (Fig. 1), the first which meets the eye when the

[1] My description is beautifully illustrated by specimens obtained from bodies prepared by injections of formaldehyde (5 per cent. of a 40 per cent. solution).

chest and pericardium are opened, is triangular in shape, slightly convex, and is directed forward and a little upward, being in sagittal mesial section parallel with the sternum. It includes the greater part of the right ventricle, and portions of the left ventricle, left auricular appendix, the whole right appendix, and part of the right auricle. It is bounded below by the sharp, almost straight antero-inferior border (margo acutus), on the left by the oblique, convex, and slightly rounded left

FIG. 1.



Anterior surface of the heart as seen in situ when the pericardium is opened.

anterior border (the left border of clinicians), and on the right by the nearly vertical and convex right anterior border (the "right border" of clinicians). Its superior angle marks the anatomical apex, and here the surface merges in the anterior walls of the aorta and pulmonary artery. Its left anterior angle forms the clinical apex.

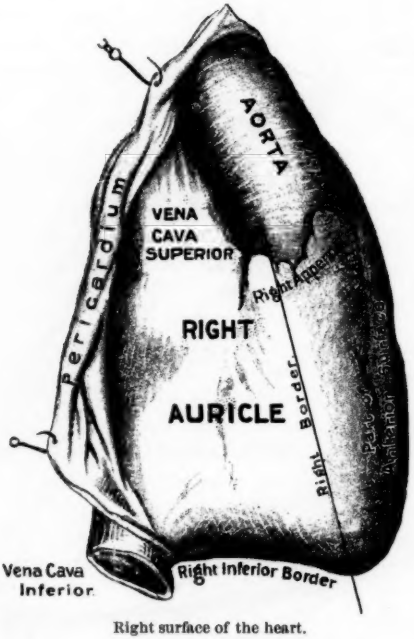
On this surface are seen the anterior or right coronary artery in the anterior auriculo-ventricular groove, while its marginal branch runs

along the antero-inferior border; and in the anterior interventricular groove is the descending branch of the posterior or left coronary artery, accompanied by the great cardiac vein.

Relations. Separated from it by the pericardium are the margins of the lungs and pleuræ, the sterno-pericardial ligaments, triangular sterni, internal mammary vessels, and sternum.

The right surface (Fig. 2) is markedly convex, four sided, lies almost vertically, and is directed toward the right. It includes the greater part

FIG. 2.



Right surface of the heart.

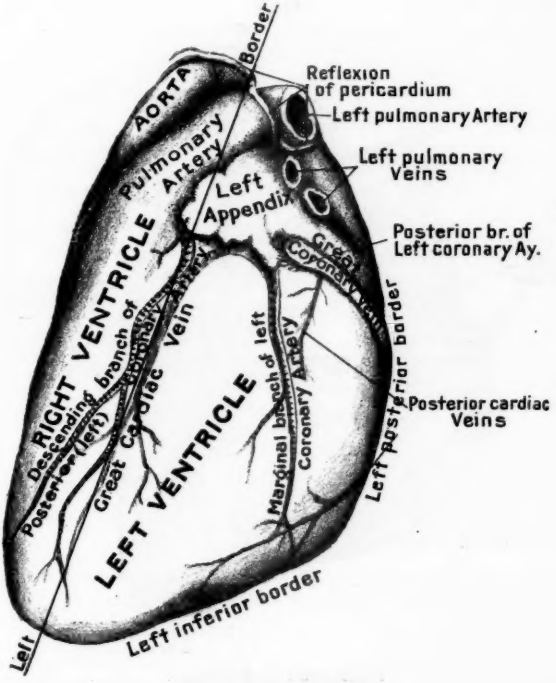
of the right auricle. Its anterior, posterior, and inferior borders are only slightly rounded, and are therefore fairly well defined. At its superior extremity the surface blends with the wall of the superior vena cava, and at its posterior inferior angle it is similarly related to the vena cava inferior.

Relations. It is separated by pericardium from the right phrenic nerve and vessels, pleura, and inner surface of the right lung.

The left surface (Fig. 3) is a convex, triangular area, directed mainly upward and toward the left. It includes about one-half of the free surface of the left ventricle and the left auricular appendix. It is

separated from the anterior surface by the left border; from the inferior surface by a rather sharp left inferior border, and behind it is bounded by the left pulmonary veins and left auriculo-ventricular groove with the coronary vein embedded therein. It presents the proximal extremities of the descending branch of the left coronary artery and great cardiac vein, the marginal and transverse branches of the same artery, and the posterior cardiac and coronary veins.

FIG. 3.



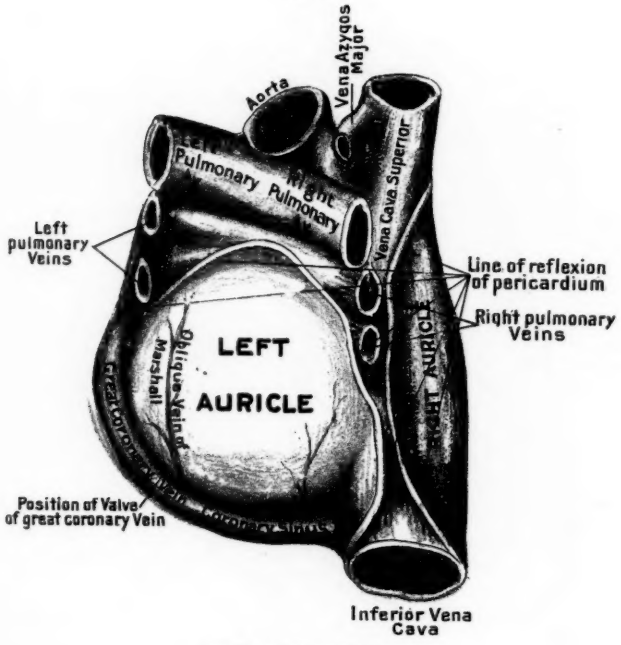
Left surface of the heart.

Relations. It is separated by pericardium from the left phrenic nerve and vessels, left pleura, and inner surface of the left lung.

The posterior surface (Fig. 4) (dorsal surface) is called the base in text-book descriptions. It is four-sided, rather narrower above than below, is convex, vertical, and directed backward. It is formed by the left auricle and by the portion of the right auricle which joins the two venæ cavæ behind, and is bounded by rather sharp and well-defined borders. It is bounded below by the inferior vena cava and the coro-

nary sinus, above by the right pulmonary artery, on the right by a fairly defined border joining the two venæ cavæ, and on the left by the cardiac openings of the left pulmonary veins and by the great coronary vein. It presents the openings of the coronary veins (right and left), the great coronary vein and coronary sinus, and the oblique vein of Marshall, which last runs downward over the surface to enter the left extremity of the coronary sinus. It is only partially invested by the serous layer of the pericardium.

FIG. 4.



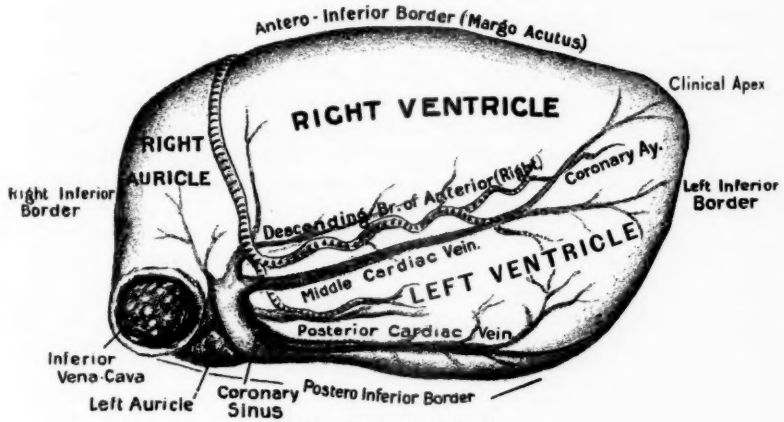
Posterior surface of the heart.

Relations. It is separated by pericardium from the bronchi, œsophagus and vagi, descending aorta, vena azygos major, and thoracic duct.

The *inferior surface* (Fig. 5) (diaphragmatic surface or *base*) is quadrilateral, slightly convex or almost flat when the ventricles contain blood, slightly concave when they are empty and relaxed. It is directed downward and a little backward and toward the right, and is bounded by rather sharp and well-defined borders. It is formed by a small portion of the right auricle and opening of the vena cava inferior, the rest of the surface being about equally divided between the right and left ventricles.

Behind the vena cava is seen a small portion of the left auricle. In addition to the inferior caval opening, it presents the inferior extremities of the right (anterior) and left (posterior) interventricular grooves, with the right coronary artery embedded in the former and the coronary sinus in the latter. Crossing it diagonally is the inferior interventricular groove, with the descending branch of the anterior (or right) coronary artery and middle cardiac vein. The posterior cardiac vein runs along its posterior border.

FIG. 5.



Inferior surface of the heart.

Relations. It is separated by the central tendon of the diaphragm and some diaphragm muscle from the superior surface (impressio cardica) of the liver.

The *apex* of the pyramid is formed by the aorta (Fig. 1), pulmonary artery (Fig. 3), and superior vena cava (Fig. 2), and these structures spring from the heart on a level with the upper margin of the third costo-sternal articulation, extending an inch and a half to the left and one inch to the right of the middle line. This we may conveniently name with clinicians the upper limit of the heart.

SUPERFICIAL INDICATIONS. The *upper limit* of the heart has just been indicated. The left half of this line will mark the position of the pulmonary and aortic valves.

The *clinical apex* is indicated by a point between the fifth and sixth ribs, three and one-half inches to the left of the middle line.

The *antero-inferior border* is to be indicated on the chest-wall by an oblique line, slightly convex downward, extending from the clinical apex on the left across and slightly upward to a point one inch to the

right of the middle line at the level of the sixth chondro-sternal articulation. Along this line the cardiac blends insensibly with hepatic dulness.

These lines being drawn, the right and left borders of the heart's anterior surface or the absolute lateral limits of the heart's dulness will be defined by convex lines joining respectively their right and left extremities. Thus, on a level with the fourth chondro-sternal articulation, the area of the heart's dulness extends three inches to the left and one and three-quarters inches to the right of the middle line.

It would seem more consistent with the above description to change some other elements in the cardiac nomenclature. For instance, the interventricular grooves are seen to be superior and inferior. The *right coronary* artery I would name *anterior*, and its branches respectively infundibular (as at present), right ventricular (now "marginal"), and inferior interventricular (now descending); the left coronary artery would be better named posterior, and its branches superior interventricular (now "descending"), left ventricular (now marginal), and auriculo-ventricular (now transverse).

RELATIONSHIP OF MIGRAINE TO EPILEPSY.

By B. K. Rachford, M.D.,
PROFESSOR OF PHYSIOLOGY AND CLINICIAN TO CHILDREN'S CLINIC, MEDICAL COLLEGE OF OHIO, CINCINNATI, OHIO.

All writers on this subject are agreed that migraine and epilepsy are kindred diseases, and that this kinship is so close that these diseases are not infrequently twin inheritances from the same neurotic ancestors.

Concerning this relationship, Landon Carter Gray says:[1] "Some eleven years ago I called attention to the association of epilepsy with migraine, not knowing till some time later that Tisset, Parry, and Liveing had previously observed the same association. In these cases epilepsy alternates with migraine, the migraine disappearing when the epilepsy appears, and the epilepsy returning when the migraine disappears. By this I do not mean to say that all cases of migraine are subject to epilepsy, but I do mean to say that there is a very close relationship between migraine and epilepsy, and in some cases the relation is so close as to permit of this alternation; indeed, almost all cases of migraine will be found at some period of their lives to have had a loss of consciousness, with or without convulsive movements, although generally this fact is strenuously denied."

On this same subject B. Sachs[2] speaks as follows: "The relation of

[1] Nervous and Mental Diseases, 1895.
[2] Nervous Diseases of Children, 1895.

migraine to epilepsy makes it also more probable that some primary peculiarity of the cortical cells is responsible for this painful affection. The resemblance is so close, and the sequence of the two diseases is so striking, that the two forms of disease may possibly represent a different degree of affection of the cortical structure."

In this connection A. Eulenburg[1] may thus be quoted: "In those epileptics who are predisposed by inheritance and constitution, attacks of migraine are among the commonest accompaniments of developed epilepsy both in early and in later years; and that in families which have tendencies to constitutional neuropathic diseases there are often individual members who suffer with migraine while others are attacked with epilepsy."

In the above quotations I have clearly and briefly set forth, in language not my own, that the kinship of migraine and epilepsy is one of the facts and not one of the theories of medicine, that I may at once follow the intent of this paper, which is to point out the manner of this relationship.

In the beginning of this study it is necessary that one should have and hold in mind a clearly defined clinical picture of both migraine and epilepsy, since these terms, in the present state of our knowledge, have a clinical rather than a pathological or etiological significance.

MIGRAINE is a name which has been given to a group of paroxysmal nervous symptoms which recur, as a rule, at irregular intervals. This symptom-group which we call migraine, *regardless of its etiology*, embraces the following more or less characteristic symptoms, viz.: Recurring paroxysmal headaches, commonly unilateral and associated, as a rule, with nausea. These headaches are self-limited, and not infrequently subside into a narcosis which produces a sleep from which the patient awakens refreshed and free from pain.

EPILEPSY is likewise a name which has been given to a group of paroxysmal nervous symptoms which recurs, as a rule, at irregular intervals. This symptom-group, which we call epilepsy, *regardless of its etiology*, embraces the following more or less characteristic symptoms, viz.: Recurring convulsive seizures, more or less general, and commonly accompanied by temporary loss of consciousness, loss of pupillary reflex, and dilatation of pupils. These convulsive seizures are self-limited, and commonly subside into a narcosis that produces a sleep from which the patient awakens convalescent from the attack.

It will also be well to hold in mind that in both migraine and epilepsy we may have among the early symptoms a slow pulse with high arterial pressure, visual disturbances, and temporary aphasia.

A clear conception of the etiologies of the symptom-groups here pre-

[1] Ziemssen's Cyclopedia, 1877.

sented is absolutely necessary to the full understanding of the relationship which exists between migraine and epilepsy. If we wish to make out the manner of their kinship we must know their antecedents, we must know from what they descended; in short, we must have their family tree. While, at the present time, this desideratum is not attainable, yet this paper is offered in the hope and belief that we have even now sufficient knowledge of their antecedents to make out in a crude way the nature of their relationship.

Etiology and Pathology of Migraine. To show our present knowledge of this subject I shall briefly quote three of our most recent writers. B. Sachs[1] says: "Since an anatomical basis is wanting, the opportunity for theoretical speculations regarding the pathology of the disease is all the greater, and has been improved by numerous writers. There can hardly be a doubt that changes in the blood-supply of the brain or its coverings are primarily responsible for the symptoms of migraine. There is also reason to believe that the sympathetic nervous system is largely involved in this disease."

Landon Carter Gray[2] says: "The pathology of migraine is unknown. . . . There has been for many years a question as to whether it was of vasomotor origin, and an attempt was made to base this upon the coloration of the face; but as this hue varies very greatly in different cases, and as there has been no proof whatsoever of the vasomotor origin, this theory remains a pure theory, worthless for any practical purposes."

James C. Wilson,[3] 1895, says: "The essential pathological process is unknown. There are no anatomical lesions. Three principal theories have been advanced: 1. That the disease is a vasomotor neurosis. . . . 2. That it is a form of neuralgia. . . . 3. The view at present generally accepted is that of Liveing, that migraine is a fulminating neurosis, in which there are periodical discharges, or nerve storms, from sensory centres corresponding to the periodical discharges from motor centres in epilepsy."

From these quotations it is plain that we know absolutely nothing of the essential pathological processes underlying migraine, and in our search through the literature of the pathology of this condition we learn that only two propositions have found a reasonable degree of acceptance in the medical mind: 1. The sensory neurons of the cerebral cortex play some *rôle* in the pathology of these attacks. 2. Reflex irritation, starting from the eye, the pelvis, the intestinal canal, or elsewhere, may be the exciting, but not the essential, cause of a migrainous attack. Such is the meagre sum of our knowledge of the pathology of migraine.

[1] Nervous Diseases of Children.
[2] Nervous and Mental Diseases.
[3] Nervous Diseases, by American Authors.

At this point I ask attention to the contributions which I have made to the pathology of this subject during the past three years. In November, 1894, I published a paper[1] entitled "Uric Acid Leucomaïnes as Factors in the Etiology of Migraine and Kindred Nervous Diseases." In this paper I recited the history of a patient suffering from migraine, and expressed the opinion that the large excess of paraxanthin, which appeared in the urine of this patient during and immediately following an attack of migraine, was an etiological factor in the production of these attacks. In June, 1895, I continued the study of this subject under the title[2] "Leucomaïn Poisoning." In this paper it was clearly demonstrated that an excessive excretion of paraxanthin was coincident with these severe migrainous attacks, and I therein expressed the belief that the paraxanthin was the direct cause of the migraine. From that time to the present I have continued the study of this subject, with the result that I am now convinced that paraxanthin is an all-important factor in the production of true migraine. This conviction has been forced upon me by the following facts, which I have repeatedly demonstrated:

1. Paraxanthin is found in normal urine in such small quantities that its poisonous properties are lost in dilution. Salomon found only 1.2 grammes in 1200 litres of normal urine; this quantity is so minute that its presence cannot be satisfactorily demonstrated in such quantities of normal urine as can conveniently be obtained from patients. In four litres of normal urine I have repeatedly failed to demonstrate the presence of paraxanthin.

2. During the past three years I have examined the urines excreted during attacks of supposed migraine, of more than half a hundred patients, and I have to record but few instances in which I failed to find paraxanthin in great excess in this urine. From the large number of examinations I have made, I believe I am justified in saying that there can be but little doubt that paraxanthin is found in enormous excess in urines excreted during attacks of true migraine. In fact, paraxanthin occurs in such great excess in migrainous urines that I now never require more than one litre (and ofttimes get less) for demonstrating its presence, and even from so small a quantity I rarely fail to demonstrate its presence in the "final fluid" by the physiological and chemical tests described in my previous papers.[3]

By way of parenthesis, I here wish to say that some of the migrainous attacks which I have found to be coincident with an excessive excretion of paraxanthin occurred at or near the menstrual time, and that

[1] Medical News, November, 1894.

[2] Transactions of the Association of American Physicians, 1895, and Medical Record, June 22, 1895.

[3] "Leucomaïn Poisoning," Transactions of the Association of American Physicians, 1895-96.

the headaches having such an association not infrequently ceased at the menopause. But in connection with this clinical fact it must also be remembered that the menstrual epoch is not a necessary factor in the production of paraxanthin headaches (migraine). In the same individual we may have an intermenstrual headache which is the exact counterpart of the menstrual headache, and I have also found that these paraxanthin headaches not infrequently continue after the menopause. Moreover, the same type of headache may occur in men, and in children before the onset of the menstrual function. These facts suggest that the relation of the menstrual epoch to paraxanthin headaches (migraine) is a complicated one. The discussion of this relationship is not within the scope of this paper.

3. Paraxanthin is not excreted in excess in the urines of migrainous patients at any other time than during an attack of migraine.

4. Paraxanthin does not occur in excess in the urine of patients suffering from severe forms of headache other than migraine. I have repeatedly failed to find paraxanthin in the urines of patients suffering from intestinal, influenzal, malarial, and nervous headaches, and for these reasons have concluded that the headaches of migrainous attacks do not produce the paraxanthin.

5. There is physiological evidence that paraxanthin is not formed in the kidneys during the process of excretion. Its presence, therefore, in increased quantities in the urine means that it was present in the blood in increased quantities directly before it appeared in the urine. Paraxanthin is excreted from the blood by the kidneys into the urine.[1]

6. Paraxanthin is the most poisonous leucomaïn of the uric acid group. It is, in fact, so poisonous that it would be strange indeed to learn that so large a quantity of this substance as is excreted during a migrainous attack could pass through the blood of a patient on its way to excretion without producing nervous symptoms.

7. Certain of the symptoms of experimental paraxanthin poisoning are very like those of a patient suffering from migraine. It is, of course, impossible to determine whether or not the animal experimented upon has headache. But the general nervous irritability, increase of reflexes, and late narcotism are present in both conditions.

In the light of the above propositions, it seems to me quite impossible to avoid the conclusion that paraxanthin is one of the important factors of true migraine. But, even if one concedes this relationship, the etiology of migraine still remains very obscure, since there are many difficult questions concerning paraxanthin yet to be solved. For example, we do not know how paraxanthin produces the symptom-group which we

[1] It is also excreted into the small intestine, but this manner of excretion does not concern our present study.

call migraine. Does it act directly on the nerve elements of the central nervous system, or does it act through the vasomotor nerves on the blood-supply of the brain? We do not know where paraxanthin is formed, where it is stored, and what conditions are necessary to cause its sudden appearance in large quantities in the blood, nor do we know how reflex and other factors act in precipitating an attack of migraine. These and many other things concerning the etiology of migraine we do not know. Yet I must insist that our ignorance concerning these important questions is not a sufficient reason for our not accepting the one proposition concerning the etiology of this disease which I think has been satisfactorily demonstrated, viz.: *Paraxanthin is an important factor of true migraine.*

Pathology of Epilepsy. The term epilepsy is used much more loosely in medical literature than is the term migraine; it covers, in fact, all sorts of convulsive seizures, be they ever so slight, and embraces, therefore, a number of pathological conditions which will, no doubt, in time be separated and classified. When this classification is made it will, I think, be found that only one form of this so-called epilepsy is closely allied to migraine, and that all the other forms are so related only as they are to chorea, hysteria, and other neuroses, toward which one may be more or less predisposed by neuropathic inheritances. While at the present time it may not be possible to make a satisfactory etiological classification of epilepsies, yet the purposes of this paper demand that such a classification be attempted. For this reason, and for this reason only, the following classification is offered.

If the literature of epilepsy is agreed upon any one fact pertaining to its etiology, it is that the all-important factor in the production of epilepsy is an irritation of the motor neurons of the cerebral cortex, and upon this foundation-stone of cortical irritation I shall attempt to build an etiological classification of the various forms of epilepsy.

The irritation which causes the violent and rapid discharge of nerve force from the motor cells of the cerebral cortex (epilepsy) may be either chemical, mechanical, or reflex.

Reflex Epilepsy. In this type of epilepsy the cells of the cerebral cortex are influenced to discharge their energy by reflex excitation; but it does not follow from this that the reflex factor is the only or even the most important factor of this form of epilepsy. In truth, one may say that a lack of development of the inhibitory centres of the brain is of more importance than the reflex causes. The lack of inhibition makes it possible for the slightest reflex cause to precipitate a discharge of nerve-force, the manifestations of which we call epilepsy. While, therefore, the reflex factor may not be the all-important one, it is a necessary one, and for this reason we may class these epilepsies as reflex. Reflex epilepsy, as here defined, embraces a large portion of the so-

called idiopathic and developmental epilepsies. The lack of inhibitory development,[1] upon which this form of epilepsy depends, may be a matter of inheritance, or it may be due to malnutrition. Cases of reflex epilepsy are, therefore, not infrequently cured by a removal of the reflex cause, or by time, which allows for the development of the inhibitory centres, or by the cure of the malnutrition, but far more frequently it continues, and there is associated with it a degree of developmental idiocy due to a lack of development of higher functions of the brain. In these cases the cortical inhibitory centres remain undeveloped throughout life, and epileptic attacks continue to be precipitated by the slightest reflex excitation; it is evident that migraine is not closely related to this degenerate type of epilepsy, which I have here classified as reflex.

Mechanical Epilepsy, otherwise known as focal epilepsy, has its origin in some mechanical irritation to the cells of the cerebral cortex. Mechanical injury to the brain may result from a foreign body, a fracture of the skull, a hemorrhage, or a new growth. In either event the mechanical irritant may produce a circumscribed meningo-encephalitis, resulting in more or less degeneration of the cells of the cerebral cortex and sclerosis of the neuroglia tissue. These secondary changes explain the continuation of the epileptic paroxysms after the original excitant has been removed. It has long been known that injuries to the skull, new growths, and cerebral hemorrhages are responsible for a large number of epilepsies; but a new interest has been added to this subject by the admirable clinical studies of B. Sachs, who demonstrated that many obscure epilepsies developed in childhood are focal epilepsies, having their origin in cortical hemorrhage which occurred in infancy. Enough has been said to show that mechanical epilepsy, apart from its classification, does not interest us in this study, since it is in no way etiologically related to migraine.

Toxic Epilepsy. There is a type of epilepsy which may be classed as toxic, the essential factors in the production of which are toxins produced in the body. Some form of auto-intoxication is, therefore, the prime factor in the production of all toxic epilepsies. Herter and Smith, in a very clever research,[2] have attempted to show that certain poisons formed in the intestinal canal may be important factors in the production of epilepsy. But our knowledge of the toxic epilepsies is so indefinite that it will not profit us to attempt a general consideration of this subject. I shall, therefore, ask attention only to one form of toxic epilepsy, namely, paraxanthin epilepsy. It is my belief that a not inconsiderable proportion of the so-called idiopathic epilepsies are produced by the irritating or toxic action of chemical products formed

[1] I noted the importance of lack of inhibition as a predisposing factor in producing epilepsy. "The Neuroses of Childhood," 1895.

[2] New York Medical Journal.

within the body, and that they should, therefore, be classified as toxic epilepsies. However this may be, I am quite sure that there is one form of toxic epilepsy which is caused by the action of paraxanthin on nerve centres, and it is to this form of epilepsy that I would ask especial attention.

In May, 1894, I published a careful study[1] of a case of epilepsy, in which I demonstrated that the urine passed by this patient immediately following an epileptoid attack contained an enormous excess of paraxanthin, and that the paraxanthin solution separated from the urine of this patient would, when introduced in the peritoneal cavities of mice, produce convulsive attacks similar to those from which my patient suffered. I also demonstrated that the urine of this patient in the interval between these attacks did not contain an excess of paraxanthin.

In November, 1894, the study of this case was continued,[2] and I therein confirmed my previous observations concerning the relation of paraxanthin to these epileptoid attacks, and further noted the following important facts: Epileptoid attacks themselves will not explain the excessive quantity of paraxanthin which is invariably found in the urine of this patient, excreted during a convulsion. This conclusion was drawn from the fact that in two cases of epilepsy, one *grand mal* and the other *petit mal*, which I carefully studied, I failed to find an excess of paraxanthin in the urines excreted by these patients during their attacks. Here, by the way of parenthesis, let me note that I have, since the publication above noted, examined the urines of many epileptics, and have always failed to find an excess of paraxanthin in the urine passed by patients afflicted with focal or with reflex (hereditary) epilepsy. It is a fact, therefore, that the epilepsy does not produce the paraxanthin, and it is also a fact that the great mass of epileptics do not have an excess of paraxanthin in their urine.

In May, 1895,[3] I concluded the study of this case in the belief that I had demonstrated, as clearly as could be demonstrated without experimentation on the human being, that paraxanthin was an essential factor of the epileptic paroxysms from which this patient suffered.

During the past three years I have examined the urines of a large number of epileptics, and have found that only a small proportion of them were suffering from *paraxanthin* epilepsy. In this study I have been impressed by the fact that it is almost impossible to make a diagnosis of paraxanthin epilepsy from the other forms of epilepsy of the *grand mal* type, from the clinical pictures alone. The urine examination is absolutely necessary to a satisfactory diagnosis. Paraxanthin epilepsy, however, presents certain clinical features which are quite sufficient to make

[1] Medical News, May 26, 1894.
[2] The Medical News, November 3, 1894.
[3] Transactions of the Association of American Physicians, 1895.

one strongly suspect this type of epilepsy. For example, paraxanthin epilepsy makes its appearance, as a rule, in middle life, and always occurs in individuals who either now suffer or have suffered from migraine in early life.

Paraxanthin epilepsy also is more common in women than in men, and the attacks not infrequently occur at the menstrual period. This fact of itself is sufficient to make us suspect all menstrual epilepsies of belonging to this type. It is also important to note that paraxanthin epilepsy is not inconsistent with the greatest intellectuality, and that it is commonly associated in later life with arterio-sclerosis and the dangers consequent to this condition. But most important of all is the fact that paraxanthin epilepsy is much more amenable to treatment than the other forms of epilepsy. Its early diagnosis is, therefore, important.

Within the past two years I have had under observation a number of cases of paraxanthin epilepsy; the study of these cases has thoroughly convinced me that paraxanthin is the essential factor in the production of the epileptic attacks from which these patients suffer. From a quart of urine passed immediately following these attacks I am able to separate sufficient paraxanthin to kill a number of mice and a large-sized guinea-pig. In these experiments I have found the convulsions produced in the guinea-pig much more instructive than those produced in the mouse. When the final fluid containing the paraxanthin separated from a quart of this urine is injected into a large-sized guinea-pig, the following symptoms are noted:

1. Increased reflex excitability immediately preceding the convulsive movements.

2. General tonic spasms of all the muscles are followed by clonic spasms, producing a tetanus in which the animal soon dies.

3. Spasms of the respiratory muscles, which cause irregular and gasping respiration, commonly accompany the tonic muscular contraction which precedes the tetanus.

4. Spasmodic contraction of the muscles of the jaw and frothing at the mouth are commonly noted.

5. Involuntary passage of urine usually occurs.

6. During the convulsion the pupils are contracted, and nystagmus is occasionally noted.

7. Death occurs (apparently from asphyxia) in from one to ten minutes after the paraxanthin injection.

cease after a time, and the animal recovers after a period of prolonged stupor.

8. If less than a lethal dose be given, the convulsive movements will

These symptoms very closely resemble the symptoms which characterize the epileptic attacks of *grand mal*, and since they were produced in the guinea-pig by paraxanthin solutions separated from the urines of

patients passed immediately following epileptic convulsions of the *grand mal* type, I do not see how one can avoid the conclusion that paraxanthin is an essential factor in the production of the epileptoid attacks from which these patients suffered.

If it be admitted, therefore, as I think it must be, that paraxanthin is an important factor in the production of one form of toxic epilepsy, then we have a rational explanation of the relation which exists between migraine and epilepsy, since paraxanthin may be the essential cause of both diseases. In conclusion, I again wish to insist that migraine is not closely related to the large group of degenerate epilepsies which I have classified as reflex or developmental, and which are commonly spoken of as idiopathic or hereditary epilepsies, and I would also insist that primary mechanical or focal epilepsy differs widely in its etiology from true migraine. Migraine, therefore, bears a close etiological relationship only to toxic epilepsy, and possibly only to that form of toxic epilepsy which I have designated as paraxanthin epilepsy.

There are many questions concerning the relationship of migraine to toxic epilepsy which cannot at present be answered. For example, we do not know why paraxanthin produces migraine in one individual and epilepsy in another. We do not know why paraxanthin should produce, in the same individual, migraine in early life and epilepsy later. It may be possible that we will find the explanation to these and other questions in certain arterial changes which result in most of these patients from the long-continued irritation of this and other poisons to the bloodvessels, or it may be that other poisons may contribute directly to the production of individual groups of symptoms in different cases. But it matters little, so far as the purposes of this inquiry go, how future investigations may answer these and other questions having a bearing on the exact relationship which exists between paraxanthin poisoning and epilepsy. My purpose here is broadly to claim that the facts which I have offered justify the conclusion that paraxanthin is an essential factor in the production of migraine and also of one form of epilepsy, and that leucomaïn poisoning (including paraxanthin) is the most important etiological factor common to these two diseases, and that their kinship largely depends upon this relationship.

REVIEWS.

A Practical Treatise on Sexual Disorders of the Male and Female. By Robert W. Taylor, A.M., M.D., Clinical Professor of Venereal Diseases at the College of Physicians and Surgeons (Columbia College), New York; Surgeon to the Bellevue Hospital and Consulting Surgeon to the City (Charity) Hospital, New York. New York and Philadelphia: Lea Brothers & Co., 1897.

No one can read this book without being impressed with the fact that the work has emanated from the pen of a specialist of ripe experience, fully capable of throwing much light on what has hitherto been obscure. It contains much that is original, together with a great deal of information which cannot fail to add greatly to our rather scanty knowledge of the subjects of which it treats.

The first chapter begins by calling the attention of the reader to a fact not usually appreciated by the general practitioner—that is, to "a thorough understanding of the male sexual disorders," of which, says the author, "more knowledge is required than is generally supposed." He goes on to show that these requirements consist in a general knowledge of medicine, an understanding of anatomy, physiology, and pathology of the nervous system; practical experience in diseases of the genito-urinary system, with a familiarity of the anatomy and physiology of the sexual tract; nature and course of syphilis; all acute and chronic infective processes of the genito-urinary tract; together with a knowledge of the use of the microscope, to enable one to make an examination of the urine and the various secretions of the body.

Most of the illustrations are original, and not only add greatly to the interest of the book, but serve to make clear the points which they are intended to elucidate.

The chapter devoted to the consideration of the anatomy, structure, and physiology of the male sexual apparatus is of unusual interest, full of original reflection, differing on some points very materially from the received view of the profession on these subjects. For example, on page 38, we find the statement, following a very conclusive argument, "to my mind the chief function of the seminal vesicles is the elaboration of a peculiar mucus in large quantity, which, in coitus, by its volume and force, carries along with it, without impediment, the seminal fluid which exists in much more sparing quantity in the ampullations of the vasa deferentia." It has hitherto been believed, and generally taught, that the seminal vessels are the reservoirs of the semen, and that their secretion served the purpose of giving life to the spermatic cell.

The argument offered by Dr. Taylor to support his views regarding the function of the seminal vesicles is not conclusive; but, taken in conjunction with the results of the studies of Dr. George S. Huntington, it would appear that the semen never reaches the seminal vesicles, and

that it is extremely probable that the ideas of the profession hitherto held in reference to the functions of the organs are incorrect.

The explanation offered on page 39 of the change and escape of the seminal secretion in ill health may possibly be correct, and is certainly the most rational that has been offered. The section devoted to the consideration of the ampullations of the vasa deferentia of the ejaculatory ducts is especially worthy of consideration.

Chapters VI. and VII. are given up to the consideration of symptomatic and atonic impotence. They are disappointing, for, while the writer has pointed out the complications which are so frequently associated with these conditions of sexual exhaustion, their consideration is too brief to be practically of service to the profession. When the great frequency of this class of cases is taken into consideration, a feeling of disappointment is experienced that the whole subject has been dismissed so summarily.

It is to be regretted that the writer has not gone more fully into a consideration of the symptoms and of the treatment of these very common and distressing afflictions. It is suggested, too, that an addition of the clinical histories of this variety of sexual disorder would add greatly to the interest of the work, and at the same time prove instructive to the general practitioner.

The article on organic impotence is complete and full of instruction.

A chapter is devoted to new growths and hypertrophies of the vulva, the subject being exhaustively treated; these conditions are familiar to genito-urinary surgeons, but have never been systematically classified nor fully described.

The remainder of the work is devoted to the consideration of sterility, masturbation, sexual excess, spermatorrhœa, sexual worry, hypochondriasis, sexual neurasthenia, conjugal onanism, priapism, sexual erethism, sexual perversion, and sterility in the female.

While the literature of medicine abounds with monographs on hypotropic ulcerative lesions of the vulva, due to syphilis, lupus, tuberculosis, and chronic ulcerative processes, the text-books contain little, if any, information on these subjects. The portion of the work devoted to the consideration of these conditions may be read by the profession with interest and instruction.

The work is written in the well-known clear and concise style of the author, and is replete with new ideas on the subjects of which it treats. It will fill a long-felt want, and should be studied, not read, by every physician who makes diseases of the sexual organs a specialty.

O. H.

A System of Medicine by Many Writers. Edited by Thomas Clifford Allbutt, M.A., M.D., F.R.C.P., F.R.S., F.L.S., F.S.A. Vol. III., pp. 1176. New York: The Macmillan Company. London: Macmillan & Co., Ltd., 1897.

This volume, which treats of infective diseases and toxicology, is the work of thirty-one different writers, who have produced a most valuable addition to medical literature, reflecting great credit on themselves and on the editor. The main divisions are (1) infective diseases of chronic

course; (2) diseases of uncertain bacteriology; (3) infective diseases communicable from animals to man; (4) diseases due to protozoa; (5) intoxications, and (6) internal parasites.

While in general it may be stated that every department is most fully and interestingly considered, certain chapters deserve especial mention.

The chapter on Tuberculosis, by Dr. Sidney Martin, treats fully of the bacteriology of the disease, the lesions produced by the bacillus and the retrogressive changes, gives most interesting details of inoculation and feeding experiments, discusses modes of infection in animals and in man, sources of infection, preventive measures, immunity, etiology, diagnosis, and other considerations.

Dr. P. S. Abraham gives a history of Leprosy, the geographical distribution of the disease, its symptomatology, pathology and bacteriology, its contagiousness, the various considerations bearing upon the question of heredity, and the influence of diet, poverty, race, climate, and segregation. Treatment is considered under three heads: hygienic, medicinal, and surgical. The whole chapter is one which cannot fail to interest the physician and the layman as well.

The chapter on Actinomycosis, by Dr. T. D. Acland, gives evidence of the author's wide reading and complete knowledge of the literature of the subject. Measles and other maladies coming within the category of "children's diseases" are well covered by the several writers, and the same may be said of smallpox and constitutional syphilis.

It would, perhaps, appear somewhat sweeping to assert that the chapters on topical diseases of uncertain bacteriology, such as typhus, dengue, yellow fever, beri-beri, epidemic dropsy, Delhi boil, etc., are individually better than can be found elsewhere in any work of a similar nature; but it may be said, without fear of successful contradiction, that collectively they may, perhaps, be equalled but not surpassed.

Vaccinia is very thoroughly presented in a chapter of 132 pages, by Drs. T. D. Acland and S. M. Copeman and Mr. Ernest Hart. Dr. Acland's connection with the Royal Commission on Vaccination for a period of seven years has given him exceptional opportunity for the clinical study of vaccinia, and Mr. Ernest Hart's lifelong connection with public health matters has abundantly qualified him for a discussion of vaccination as a branch of preventive medicine.

Simply to state that the somewhat brief chapter on Malarial Fever is the work of Dr. William Osler is a sufficient guarantee of the excellent manner in which that subject is handled. Dr. H. A. Lafleur writes on Amœbic Dysentery, which has been a subject of some original research by himself in connection with others.

The chapters on Intoxications, covering poisoning by ptomaïns, grain, mushrooms, snake-bite, etc., and alcoholism, commend themselves particularly to the sanitarian and therapeutist.

Dr. Allbutt's chapter on Opium Poisoning and other intoxications is exceptionally interesting and valuable, and his remarks on the treatment of poisoning by opium may well be taken by writers on therapeutics and toxicology as a basis for revision of much that is written on that subject. Occasional flashes of a certain grim humor add more or less to the pleasure of perusal.

Dr. Thomas Oliver's work on metallic and some other forms of poisoning is a valuable contribution to industrial hygiene, though his chemistry is sometimes not beyond criticism.

A hundred pages on Internal Parasites, by Drs. Griffiths, Manson, Guillemard, Verco and Stirling, complete the volume in a most highly satisfactory manner, presenting the subject very fully and with many instructive illustrations. C. H.

INCOMPATIBILITIES IN PRESCRIPTIONS, FOR STUDENTS IN PHARMACY AND MEDICINE AND PRACTISING PHARMACISTS AND PHYSICIANS. By EDSEL A. RUDDIMAN, Ph.M., M.D., Adjunct Professor of Pharmacy and Materia Medica in Vanderbilt University. Pp. iv. 264. New York: John Wiley & Sons, 1897.

THE first part of this volume contains in convenient and condensed form the more common incompatibilities; the second, a list of incompatible prescriptions to the number of three hundred and twenty-five. Following this are explanatory notes which point out the difficulty and give the best means of avoiding or overcoming it. On page 240 is a table showing the effect of rubbing together equal weights of two solids. There are two indexes—one to incompatibilities and the other to prescriptions. That such a book is needed by the physician the prescription-files of any pharmacy would demonstrate, and that the pharmacist could profit from its study the frequent, unnecessarily unsightly bottles sent out from the shops are evidence. In examining five hundred consecutive prescriptions from an excellent pharmacist, written by one hundred and eighteen physicians, we recently found chemical incompatibility in 7.2 and physiological in 6.2 per cent. We all can improve our prescribing by studying this book. How many syphilographers, for instance, are aware that corrosive sublimate is slowly converted into calomel by the compound syrup of sarsaparilla (p. 63), or that milk-sugar when triturated with calomel changes some of it into corrosive sublimate (p. 65)? As to the correctness of the statements contained in the book the author frequently gives his authority and often shows that he has verified the observations of others. We doubt very much, however, if morphine is physiologically incompatible with paraldehyde (p. 78), since respiratory paralysis is a marked feature in poisoning from either. The author is to be congratulated upon his rigid adherence to modern chemical and pharmacopœial nomenclature—a custom more honored in the breach than in the observance. But we prefer alcoholic potassium hydrate (p. 43) to alcoholic potash (p. 46 and elsewhere), and alkaline rather than alkali hydrates. Typographical errors are extremely rare—picrotexin (p. 63) is an example. The volume exhibits the result of patient research and long-continued industry, and its appearance should be rewarded by better work by the physician as well as by the pharmacist.

R. W. W.

VADE-MECUM OF OPHTHALMOLOGICAL THERAPEUTICS. By DR. LANDOLT and DR. GYGAX, of Paris. 16mo., pp. vi. 138. Philadelphia: J. B. Lippincott Company, 1898.

THE statement of the authors that "This work is not a treatise of ophthalmological therapy, nor is it a dictionary," indicates that they feel that their work borders upon both of these.

Its contents are divided into paragraphs arranged according to topics in alphabetical order. This obviates the need of an index, but leads to the necessity of many cross-references and to occasional repetitions. Thus, under the heading "Cornea, foreign body of the," we find it stated, "Where foreign body has penetrated into the tissue of the iris: iridectomy," and practically the same thing under "Iris, trauma of the," while "Foreign body" is not given as a topic.

In most respects the teaching is sound, clear, and as complete as it could possibly be made in the space allotted. The three pages devoted to "Specific Treatment, Antisyphilitic Treatment," for which the authors express their thanks to Prof. Fournier, contain a most admirable résumé of the subject. In regard to the treatment for hyperopia and myopia, however, the attitude seems to be: If the patient must wear glasses, let them give him as little help as he will be at all satisfied with. For hyperopia they say: "For distant vision correct at most but the manifest hyperopia." Only for aphakia do they advise the glass correcting the entire error. Again we quote: "Patient hardly ever tolerates permanently the glass completely correcting this myopia." "It is nearly always better for a myope to use no concave glasses for near vision."

Such an attitude toward these forms of ametropia is responsible for much unnecessary suffering, and, in so far as it is maintained, will place the work of the ophthalmic surgeon below that of the optician who is willing to sell his customer whatever glasses the customer chooses. It is a survival of the fear of correcting lenses, an error prevalent in the profession before the time of Donders, and which exact study of the subject of refraction should ere this have dissipated. Inability to recognize the few defects of this kind may cause the book to do some harm among those not widely read on the subject, but on the whole it will be useful.

It is a work that deserves and will have a wide circulation among general practitioners, and may also be read with great benefit by the specialist. It is neatly printed in convenient form to carry in the pocket, and the arrangement of matter and selection of type are such as to make its contents quickly available to any one. E. J.

A Text-Book on Mental Diseases. For the Use of Students and Practitioners of Medicine. By Theodore H. Kellogg, A.M., M.D. New York: William Wood & Co., 1897.

The work before us is a large volume of seven hundred and fifty-nine pages. The object of the book, as stated in the preface, is "to set forth in a condensed but comprehensive manner the present state of the science of mental diseases. The book is made to embrace the wide range of the history, statistics, nosology, etiology, clinical course, symptomatology, pathology, diagnosis, prognosis, and treatment of mental disorders. An attempt has been made to introduce such clear and systematic subdivisions as would best tend to facilitate the comprehension of the whole subject and render the book available for students and practitioners of medicine. The book aims to be a practical guide to the diagnosis and

treatment of all the various types of insanity with which the physician has to deal in public hospitals or private practice, and also to act as a work of ready reference to psychiatrists in the emergencies of their specialty."

The subject is divided into two parts, the first of which deals with "general mental pathology," and the second "the special groups and typical forms of insanity." Part I. embraces a series of interesting and well-written chapters. Among them are to be specially mentioned one on the Nosology of Insanity and those of Psychical and Somatic Symptomatology. In that on nosology the subject of classification is considered in a very systematic manner.

However, general statements, generalizations, and time-worn platitudes are of little value when compared with the specific statements which should be made regarding the etiology, symptomatology, diagnosis, and treatment of each specific mental affection. In the experience of the writer these general sections in treatises of mental diseases are rarely, if ever, read by the student or by the practitioner. What the student and physican need are systematic statements and elaborate clinical descriptions of the actual diseases with which they meet in daily life. It is, therefore, disappointing to find that while 516 pages are given up to the consideration of merely general matters, only 241 are given to the actual discussion of the various forms of insanity. The conviction forces itself upon the reader that the special and clinical part of the book must have been condensed and much abbreviated in order to admit of the expansion of the almost useless general section. The writer does not desire to imply that the general section is badly written, but merely that it is disproportionately large and contains much matter that has no practical application. In detail our criticism of the chapters is somewhat as follows:

Chapter I., on the History of Insanity, is sufficiently condensed; that on Statistics of Insanity had best been entirely omitted, while that on the Nosology of Insanity is an admirable review of the various forms of classification at present adopted; but the author errs, in the opinion of the writer, in not contenting himself in the present unfortunate state of our knowledge by simply *enumerating* the various forms in which insanity manifests itself. The chapter on the etiology of insanity could have been much condensed, while that on the evolution, stadia, clinical progression and termination of mental disorders could with advantage have been entirely omitted. The chapters on Psychic and Somatic Symptomatology are excellent and deserve unstinted praise, and yet it is an open question whether the student will not gain a clearer conception of the symptomatology of insanity in general by a study of the *symptomatology of the specific forms.* In the opinion of the writer this is the only way in which a knowledge of the symptoms is really acquired, especially as insanity is a disorder a knowledge of which can only be acquired by the actual study of living cases. The generalizations present in the general sections of works on insanity are abstractions of clinical experience, and are not materials fit for presentation to the student or practitioner save to an exceedingly limited degree. The practical value of chapters on the Pathology, Diagnosis, Prognosis, and Treatment of Insanity are also, in the opinion of the writer, equally open to question. Certainly in the consideration of each specific form of mental disease every factor considered in Part I. from a general stand-point

must be considered a second time in the special section, and it is specific statements of facts in relation to specific affections that are of importance. Considered in this light, it is certainly an anomaly that more than two-thirds of the book should be devoted to general considerations and less than one-third to detailed descriptions of the various mental diseases. It is difficult to avoid the inference that as a result of this arrangement the special section has suffered, and an examination of its pages shows this to be the case. The descriptions are for the most part exceedingly condensed. For instance, melancholia is considered in less than six pages; mania in less than eight, while paresis, which is a very world within itself, is disposed of in twenty-two pages—that is, history, etiology, symptomatology, diagnosis, prognosis, and treatment. The other forms of insanity are considered in the like condensed manner.

The author classifies the various forms of insanity into two groups:

Group A "is made in accordance with the etiological and pathological principles, and is, hence, briefly defined as etio-pathological, and as having assignable etiological and pathological relations." It consists of seven classes: first, insanity from general arrested development; second, insanity emerging from constitutional neuropathic states, usually hereditary, though occasionally acquired; third, insanity with established neuroses; fourth, insanity in connection with the physiological crises; fifth, insanity with general systemic morbid states; sixth, insanity with definite pathological conditions of the encephalo-spinal, vasomotor, or peripheral nervous system, and seventh, insanity from pathological psychic influences.

The second group "consists of the simple psychoses without definitely assignable etiological and pathological relations, and hence the group is briefly named psycho-symptomatological." It consists of the emotional (Class VIII.), the intellectual (Class IX.), and volitional (Class X.) insanities. In the present unsatisfactory state of our knowledge of insanity, almost any classification can be adopted, and a reviewer could reasonably be charged with being captious if he found fault with the classification adopted by any one author. As a matter of fact, the classification—that is to say, the order in which the subjects are considered—is of a relatively slight importance, the real point of importance being the accuracy, the fulness and completeness of the clinical descriptions. A standard work upon insanity should consist of elaborate and comprehensive word-pictures of the things actually met with in the daily life of the physician in or out of the asylum, and should be interspersed with appropriate records of cases actually studied and observed. This is assuredly not the case in the special section of the work before us.

F. X. D.

THE NORMAL AND PATHOLOGICAL CIRCULATION IN THE CENTRAL NERVOUS SYSTEM. ORIGINAL STUDIES. By WILLIAM BROWNING, Ph.B., M.D. Philadelphia: J. B. Lippincott Co., 1897.

DR. BROWNING'S book is made up chiefly of original papers which have been read before various scientific societies and published in various medical journals. It is, therefore, rather lacking in unity of design and systematic presentation of its subject, and these are defects for which the reader, unfortunately, suffers more or less all the way

through its pages. The author's commendable plan, however, has been to present a series of articles which should embody something original, and, even if he has done this at the sacrifice of a systematic scheme, he may reasonably claim the indulgence of his readers.

The first six articles deal exclusively with anatomical subjects, and are based on some original experimental work. The first paper is on the important subject of the spinal efferents for the cerebro-spinal fluid. Browning concludes that outlets for the cerebro-spinal fluid exist along the lumbar nerves in the lower animals at all ages, but in the human being only during intra-uterine life. This has been shown in the dog and in the human fœtus by the deposit in the psoas muscle of material injected into the subarachnoid space. The obliteration of these efferents in the human infant begins, according to the author, at or about birth, and constitutes one of the earliest changes of extra-uterine life. In the place of the spinal efferents the arachnoidal villi, and perhaps the other cranial outlets, increase as life advances, and act as substitutes. With reference to these general conclusions, we may say that the paper does not seem to us to be entirely convincing, and as it deals but little with the cranial efferents its contribution to our knowledge of the circulation of the cerebro-spinal fluid is negative rather than positive. The other anatomical studies in the book have largely to do with the disposition of the veins of the brain, and especially with reference to the theory of the developmental rotation of the brain. In these sections we sadly miss good diagrams, without which anatomical studies of the brain are exceedingly difficult to utilize.

The most important clinical study in Browning's book is that on hydrocephalus. The chapter on Obstructive Hydrocephalus and the mechanical principles on which its development depends is especially noteworthy. The author discusses the various theories that have been offered to account for the increase of fluid. These are (1) the closure of the foramen of Magendie, causing retention of fluid; (2) obstruction of the veins of Galen, causing over-production of fluid; (3) inflammation or irritation of the choroidal villi in the ventricles, such as ependymitis, tubercular meningitis, etc., also causing over-production; (4) abnormally diminished resistance in the cranial walls, in very young, rickety, and syphilitic subjects; and (5) obstruction of the ultimate absorbents in the subarachnoid space. This last cause is considered by the author to be an active one, in accord with his belief that in the very young the cranial absorbents have not yet assumed full physiological importance, and hence may be more readily obstructed than later. Browning reviews the literature to prove that obstruction may also act at the several outlets of the ventricles (foramen of Monro, aqueduct of Sylvius, and foramen of Magendie) and also upon the venous system (veins of Galen, etc.) to cause hydrocephalus. The proof is, of course, conclusive so far as it goes; but it seems to us to fall far short of demonstrating that gross obstructive lesions at these several points are either common or essential in this affection. Again, in cases of obstruction of the outlets of the ventricles, as from gross inflammatory or other lesions, it must often be difficult to determine positively that the venous system also has not been interfered with, and hence the claim for a pure type, dependent alone on interference with the circulation of the cerebro-spinal fluid, must still remain undecided. In the treatment of hydrocephalus the author finds more to hope for from surgical interference than we

can. The operation for opening and draining the fourth ventricle (by trephining the occipital bone, lifting up the cerebellum, and even attempting to clear away obstructions at the foramen of Magendie) seems to us to be a particularly rash procedure, especially in a child that may be suffering with tubercular meningitis or some other form of infectious brain-disorder. Anyhow, our data are rather too limited as yet to permit us to diagnosticate accurately obstructive lesions at the foramen of Magendie (the metapore), and, considering the uncertainty of such a lesion being a frequent factor in hydrocephalus, or even an exclusive factor when it is present, our judgment and our practice should still be conservative in the extreme. The same may be said of Quincke's operation of lumbar puncture. It does not go to the seat of trouble, especially if the obstruction be at any point within the ventricles, and, as in cases of cerebellar tumor, as Browning points out, it may not be unattended with risks.

Other important chapters in Browning's book are on double and symmetrical hemorrhages in the brain, and various types of apoplexy. The final chapter, dealing with the early treatment, based on the differential diagnosis, of hemorrhage, embolism, thrombosis, etc., is a praiseworthy and suggestive paper devoted to the amelioration of a class of cases in which treatment is too often discouraging and even futile. On the whole, we can recommend the book as a thoughtful and scientific, though somewhat desultory, work in a most important field of pathology. J. H. L.

PATHOLOGIE UND THERAPIE DER MÄNNLICHEN IMPOTENZ. Von DR. VICTOR G. VECKI V. GYURKOVECHKY. Zweite, vollständig umgearbeitete Auflage. Pp. 239. Wien und Leipzig: Urban und Schwarzenberg, 1897.

IMPOTENCE IN THE MALE. Second Edition.

WE have read this volume, which is mostly devoted to the pathology of impotence, with considerable interest. The author has industriously read the extant English, German, French, and Italian literature bearing upon the subject, and apparently has given credence to the greater portion of that read. Naturally, with the exception of a limited number of personal observations here related, we expect a compilation of others' opinions based upon more or less accurately observed facts. The book is plain-spoken, and rather more importance is given to the subject than seems necessary. In his therapeutics the treatment must, of course, depend largely upon the causes of the difficulty, and these have been thoroughly gone over in the pathology. In drug-treatment the author cites remedies usually to enter objection to their use. The main reliance seems to be placed on hydrotherapy, less upon electricity, and little upon hypnotic suggestion. It is scientific in tone, possibly sufficiently popular in style to be dangerous, but on the whole quite satisfactory and readable. The author is modest in his case-reporting. For the physician who wishes to know the literature of the subject this volume will be very acceptable. R. W. W.

PROGRESS

OF

MEDICAL SCIENCE.

THERAPEUTICS.

UNDER THE CHARGE OF

REYNOLD W. WILCOX, M.D., LL.D.,

PROFESSOR OF MEDICINE AND THERAPEUTICS AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL; VISITING PHYSICIAN TO ST. MARK'S HOSPITAL.

The Use of Methyl Salicylate for Rheumatism.—DR. GILBERT LASSERE, noting the disagreeable odor to which this drug gives rise when used externally, determined to obtain the remedy so that it could be administered by the mouth. He makes use of the pure synthetic product in the following formula: Methyl salicylate, 1; punch (sweetened), 100; distilled water, 100. Six ounces are taken during the twenty-four hours in tablespoonful doses, and this has been sufficient to relieve the pain in forty cases which were about equally divided between the acute and subacute varieties. Salicyluric acid was found in the urine witin two hours from the administration of the drug. It is also eliminated by the skin. The drug appears to be useful also in gout, but since two cases only have been under observation definite statements cannot be made.—*Les Nouveaux Remèdes*, 1897, No. 22, p. 683.

The Treatment of Pernicious Malaria.—DR. CLARENCE J. MANLY produces cinchonism as rapidly as possible, and, since the temperature variations are exceedingly irregular, larger doses are necessary. The stomach can rarely be used, and hypodermatic and intravenous injections (Baccelli's method) are to be considered. The great depression must be combated by strychnine and digitalis. The patient must be sustained by enemata of whiskey, peptonized foods, and broths.—*Therapeutic Gazette*, 1897, No. 12, p. 809.

Indications for the Use of Urotropin.—DR. L. CASPER believes that in renal colic the attacks are cut short, the dull after-pains lessened or abolished, and the recurrence of the attack is postponed. With daily doses of from fifteen to thirty grains, excellent results are obtained in phosphaturia. In nearly every instance the urine became clear, even if it had been opaque for years. In cystitis and pyelitis it acts as a disinfectant of the passages; here

the daily dose of a drachm is necessary. In ammoniacal urine the pus is diminished and the remedy shows a distinct antibacterial action, especially in elderly persons who suffer from chronic suppuration in the pelvis of the kidney; and in the bladder we obtain excellent results. These are the instances in which we fear urinary poisoning. Its bactericidal properties are readily understood when we note that in its decomposition formaldehyde is set free. Since only a part is thus decomposed and the remainder is excreted as urotropin, large doses (as above mentioned) can be safely given.—*Therapeutische Monatshefte*, 1897, No. 52, S. 1338.

[Under our observation a single instance of long-standing ammoniacal cystitis, resulting from prostatic hypertrophy in a patient whose heart and kidneys forbade operation, and an entrance upon catheter life was inadvisable, the use of this remedy in fifteen-grain doses at night gave speedy relief. The improvement has continued with the abôve dose on alternate nights — R. W. W.]

The Treatment of Chyluria by Ichthyol.—Dr. Moncorvo, Jr., has made use of various remedies in the treatment of this disease, which is common in the tropics. He mentions iron, astringents, arsenic, quinine, manganese, various vegetable remedies, and hydrotherapy; all these have failed. Recognizing the parasitic origin of the disease, turpentine, cusso, naphtol, thymol, methylene-blue, and asaprol have been tried. Finally ichthyol, a germicide and astringent, was given in dose from seven gradually increased to thirty grains per day, in pill form. Two instances are reported of its successful use.—*Les Nouveaux Remèdes*, 1897, No. 23, p. 719.

Eucasin.—Dr. Hugo Weiss notes the easy absorption, marked nutritive value, and absence of irritation of this food. As a food it is of equal value with casein, but the latter is not readily absorbed and frequently gives rise to intestinal fermentation. For rectal administration this preparation is available. In the hyperemesis of pregnancy nutritive enemata of this substance caused a constant increase of body-weight. Especial attention is called to the fact that it contains no nuclein, so that it does not increase the elimination of uric acid. Control observations upon patients suffering from the uric acid diathesis have shown that this opinion is correct. Frequently there has been noticed an increase in the amount of hæmoglobin in the blood. Since this food contains 95.65 per cent. of readily absorbed albumin, its value is readily apparent.—*Therapeutische Wochenschrift*, 1897, No. 51, S. 1326.

The Treatment of Gonorrhœa in Women.—Dr. C. F. Marshall points out the incompleteness of vaginal douches, and believes that the only forms of treatment which will reach the upper portion of the vagina and distend the rugæ are (1) applications of lotions, as silver nitrate, through a speculum to the whole of the vaginal surface; (2) plugging of the vagina with wool tampons soaked in medicated fluids; and (3) medicated pessaries, the best of which are the gelatin-glycerin. The silver-nitrate treatment cannot be carried out thoroughly without an anæsthetic, and is very irritating. For the wool-tampon method the best drug is naphtol in 2 per cent. solution in alcohol and glycerin. This, however, is irritating. A glycerin solution of ichthyol

(2 to 5 per cent.) is also good. Borax-glycerin or corrosive mercuric chloride in glycerin (1 to 500) may be used. If the inflammation is too acute a vaginal douche of zinc sulphate, lead acetate, and borax, of each one drachm; glycerin, one ounce; to one pint of water, may be used. For the gelatin-glycerin pessary: iodine 1 to 2 per cent.; ichthyol, 2 to 5; corrosive mercuric chloride, $\frac{1}{3}$ of 1; argonin, 5; lysol, 2; formalin, 2; airol, 10; naphtol, 2 per cent. solutions. The best results have been obtained with iodine and ichthyol. Urethritis is best treated with copaiba and santal. Abscess of Bartholin's gland may be cured by simple incision; but if a cyst has previously existed, it should be excised. Endocervicitis should be treated locally by copper sulphate (1 to 12) applied on a cotton-wrapped probe. If endometritis is severe the uterus should be dilated, curetted, and iodine-phenol applied over the whole surface. Condylomata, if numerous, are treated with a powder composed of copper subacetate, 1; and powdered savine, 3. Large ones may be removed by the Paquelin cautery.—*Treatment*, 1897, No. 18, p. 413.

Physiological Experiments with Intestinal Irrigation.—Dr. Robert Coleman Kemp has carried out laboratory experiments on dogs to determine the effect of continuous intestinal irrigation on the pulse tension, temperature, body and blood, renal secretion, and intestinal absorption. The irrigating fluid consisted of normal salt solution (one drachm of salt to a pint of water). His deductions are as follows: 1. Pulse tension: irrigate at 100°, 101°, 102° F., or even to 103°–104° F., if increase is to be avoided. If moderate increase is not objectionable, a temperature of 105°–108° F. can be employed. If it is desired to rapidly increase it and to stimulate the heart, irrigate with a temperature of 110° F. and increase it steadily to 120° F. This is excellent in shock and allied conditions; before or during severe operation, to prevent shock; and from the commencement of chloroform anæsthesia, to prevent the sudden dilatation of the bloodvessels. Cold is a temporary stimulant, and cold irrigation will for a time markedly increase it; later, it is a depressant, and it falls. Cold should therefore be employed with caution. Clinically, it has been noted that more patients have an idiosyncrasy to cold than to heat. 2. Shock from hemorrhage: irrigate with normal saline solution, 110°–120° F., in this condition. With the double-current method, the patient receives a continuous enema at the desired temperature, and the quantity of the fluid can be absolutely regulated by the operator. 3. Temperature: hot irrigation, 110°–120° F., when prolonged, increases the temperature of the body and blood. The increase of blood temperature is possibly due in part to the contiguity of the solution to the great vessels, as there does not seem to be a proportionate ratio of increase of temperature of the body to that of the blood if due merely to effect on heat-centres. In other words, the influence on the blood temperature seems to be greater in proportion than it does on that of the body. The heated blood would undoubtedly be of value in stimulating the heart, and this shows the further advantage of irrigation at a high temperature in shock and allied conditions. If the patient's temperature is already high, it might be dangerous to irrigate with fluid at a high temperature for fear of increasing the temperature of the patient. Cold irrigation reduces temperature, but is

depressing after twenty to twenty-five minutes. At first, cold acts as a stimulant. Clinically, cold irrigation has been employed with success to aid in the reduction of temperature in the diarrhœas of children and in dysentery. It might be employed in sthenic cases to reduce temperature, if used with caution for five or ten minutes, during which friction of the limbs would be advisable. The temperature at the start should not be below 60°–70° F. In duodenal jaundice cold irrigation (two quarts) and the alternate hot and cold douche (two quarts each) have been beneficial. 4. Renal secretion: in ten minutes irrigation at the higher temperatures, especially 110°–120° F., stimulates the kidneys to secretion by the heat and by the stimulating effect on the circulation, also by the heated blood flowing through the organs. In twenty minutes irrigation at 110°–120° F. causes excretion from the kidneys actually through absorption from the intestines. If possible, if the temperature or pulse tension is not too high, irrigate at the higher temperatures, 110°–120° F. This seems to act in a double way: by the stimulating effect on the circulation and by the heat, and also by absorption of fluid from the intestine. The lower temperatures, 100°–104° F., seem less powerful and to act through absorption. There is some increase of tension, but no increase in secretion of urine could be discovered until twenty minutes had elapsed. The slight increase of tension would not likely account for the renal secretion. If it is desired to increase renal secretion without increasing pulse tension or temperature, irrigate at 100°–104° F., and at 105°–108° F. if moderate increase is not objectionable. Intestinal absorption from the large intestine occurs in twenty minutes. This has an important bearing on self-infection, and shows the value of irrigation in diarrhœas. Clinically, hot saline irrigation is an excellent remedy in acute uræmic suppression or in cases of renal insufficiency. Cold irrigation practically inhibits intestinal absorption, on account of the effect on the circulation at the end of twenty to twenty-five minutes, and therefore should be absolutely contraindicated in renal disease, as well as the use of cold enemata.—*New York Medical Journal*, 1898, No. 1000, p. 141.

The Action of Physostigmine upon Intestinal Movements.—Dottor Gaetano Traversa, from careful experiments upon dogs and horses, reaches the following conclusions: 1. Physostigmine not only exaggerates the peristaltic movements, but also causes a violent and generalized contraction of the intestine and, finally, tetanus and contractures. If the contraction predominates in the longitudinal fibres, the intestine becomes wrinkled; if in the circular, it is beaded, ringed, or, if the contraction is violent and diffuse, ribbon-like. 2. The exaggerated movements and general contraction of the intestines cease upon the use of atropine, but they do not occur in atropinized animals. 3. The higher nerve centres have no influence upon the production of these phenomena; neither the vagus, spinal cord, nor abdominal sympathetic ganglia. 4. A loop of intestine detached from the body and kept alive by artificial circulation gives the same reaction to physostigmine as intestines in the living body. 5. The changes in motor activity do not depend upon the modification of the intestinal circulation. 6. A loop of intestine which remains inactive from the diffuse tonic contraction of its walls, brought about by large doses of physostigmine, regains its move-

ments under the influence of atropine when circulating in the blood. The spasm of the muscular wall and the narrowing of the bloodvessels disappear, and the lumen of the bowel becomes larger. 7. Physostigmine produces exaggerated peristalsis and violent and diffuse contractions of the intestine solely by excitation of the peripheral motor apparatus. 8. It is an antagonist to atropine, but it is always one-sided [see 2]. 9. So far as the intestine is concerned, the action of physostigmine is identical in intensity and duration of effect with pilocarpine, not only nosographically, but as well mechanically.—*Il Policlinico*, 1898, No. 1, S. 1.

Treatment of Thermic Fever in Infants.—Dr. John Zahorsky condemns the ice-bath, since this contracts the peripheral arterioles and markedly increases muscular rigidity. In the milder form, sponging the body with hydrant-water and the administration of more water internally is all that is required. In the severe forms a bath, the temperature of which is not below 60° F., may be used; at the same time friction should be vigorously applied to keep the peripheral arterioles dilated. Stimulants may be given as required. In the hyperpyrexial forms it is well to make the skin intensely red, as by nitroglycerin, friction with towel or hand, or a mustard bath; these increase the peripheral circulation; then even sponging with hydrant-water will rapidly produce the desired result. Spraying cold water on the patient, which mechanically causes relaxation of the arterioles and reflexly acts as a powerful stimulant, has been found to be the most effective treatment. The water should not be too cold. For convulsions and tonic spasms chloroform is important. Free perspiration should be induced as soon as possible. Diuretics act well by assisting the elimination of waste products. Unless this is done the fever will rise again, for this tendency persists for two or three days. The stimulants to be recommended are strophanthus, ammonia, ether, camphor, and alcohol. Nux vomica should not be administered, as it may only be synergist to the toxin. Water should be given as soon as possible and freely administered until convalescence.—*Pediatrics*, 1898, No. 4, p. 143.

Eugenia Jambolana.—Mr. Norman S. Rudolf, noting that there are three varieties of jambul growing in the gardens in India, states that the ripe fruit can be eaten in season. At other times the fruit preserved in spirit can be employed. The powdered seed in doses of about five to ten grains three times a day is recommended as a very effective means of administration. A vinegar of a light-pink color can be prepared by exposing the juice of the ripe fruit, contained in porcelain vessels, to the heat of the sun. After the juice has commenced to ferment it is filtered and again set in a warm place for a fortnight, when it is ready for use. It is believed that age increases the efficacy of this vinegar. The best form is either the whole fruit preserved in alcohol, the powdered seed, or a fluid extract of the seeds. The natives believe that the pulp of the dried fruit is almost worthless. Considering the high reputation which jambul has held in the East for centuries, it does not seem as though it has attracted in the West the attention which it deserves. The reason is apparently that utterly worthless lots of seed are exported and find their way into the laboratories of manufacturing chemists

in Europe and America. To obtain the seed fit for use it is necessary that the tree from which it is gathered should be of the right variety of jambul, and that no "wind-falls" or rotten fruit be included. The native physicians believe that this remedy is of the greatest use in the treatment of diabetes, and that there is no necessity to restrict the patient's diet, since this prevents the possibility of the formation [excretion?] of sugar in the urine.—*Bulletin of Pharmacy*, 1898, No. 1, p. 6.

[A recent re-investigation of this drug convinces us of its undoubted value. However, further chemical and physiological study is necessary.—R. W. W.]

The Treatment of Hæmoptysis.—Dr. C. Y. Bliss classifies this as follows: 1. Moral management. The alarm should be relieved by the assurance that it is not likely to prove fatal. Rest and quiet should be enjoined. 2. Position and rest. The patient should be left in a recumbent position and his clothing loosened without unduly disturbing him. Auscultation of the breathing over the front of the chest is permissible. If the bleeding has been profuse into one lung, an appropriate change of position may enable him to cough up the blood and avert impending asphyxia. 3. Local applications. Ice to the chest apparently does good and has a soothing effect upon the patient's mind. The writer has not observed that catarrh follows its use. 4. Relief of cough. Unless this is severe it does not need treatment. It may even be beneficial in freeing the lungs from blood. If it is desired to reduce its frequency and severity, morphine hypodermatically is to be preferred. 5. Treatment addressed to the nervous system. In cases of nervous excitement opium in some form is most desirable. 6. The action of the bowels. It is generally, not to say invariably, advisable that the bowels should be opened as soon as possible and be kept freely opened; for, since the abdominal circulation is capable of containing a large part of the total amount of blood, it is not unreasonable to believe that the induction of brisk bowel-action by means of purgatives will cause a large amount of blood to pass into the intestinal vessels, and that this may tend to reduce the tension of the circulation elsewhere. The use of the bedpan should be enjoined, but enemata should be avoided. Probably nothing is better than the immediate administration of five grains of calomel, followed by a saline purgative at an appropriate interval. After this, free action of the bowels may be maintained by adding to the mixture to be given some magnesium sulphate and a carminative, as syrup of ginger, in sufficient quantity to produce two or three watery stools daily. 7. Other medicinal remedies. Objection is made to gallic acid because of its constipating effects and to ergot because, while it contracts the smaller systemic arteries, it has no effect upon those of the lungs, and it may indirectly tend rather to raise the tension of the pulmonary circulation. There is little evidence to show that hamamelis, diluted sulphuric acid, and other astringents of this class, have any definite effect upon the small vessels of the lungs. More, perhaps, may be said for turpentine, the action of which in the hemorrhage of purpura and scurvy is recognized to be of great value. 8. Diet. First in importance is the privation of fluid. If possible, reduce the total intake of fluid to half a pint in twenty-four hours. There is no valid argument for the administration of

ice. A spare, semi-solid diet, such as a little milk pudding, bread and butter, thin sandwiches made with pounded chicken or hard-boiled eggs, which can be increased in quantity or improved in quality, should be employed. Stimulants of all kinds should be forbidden, excepting under especial circumstances.—*The Clinical Journal*, 1898, No. 271, p. 161.

The Treatment of Acute Articular Rheumatism by Intravenous Injections of Sublimate.—Dr. Gustav Singer details the process as follows: The arm is left hanging over the edge of the bed, in order to bring the veins into prominence, or a compress above the elbow is employed for this purpose. The skin in the region of the elbow is then disinfected, first by energetic rubbing with soft soap and warm water, next with a $\frac{1}{10}$ per cent. sublimate solution, and lastly with ether and alcohol. A sterilized syringe provided with an asbestos piston is filled with the solution and inserted into the lumen of the vein in the direction of the blood-current and parallel to its course. As soon as the color of the solution in the syringe has indicated that the needle has entered the vein (blood entering reddens the solution), the compress is removed and the injection is slowly made. The point of insertion is covered with the finger, the needle removed, and the opening closed with iodoform-gauze and an appropriate bandage. The solution employed is: corrosive mercuric chloride, 1 or 2; sodium chloride, 1 or 2; distilled water, 10. The dose of sublimate is from $\frac{1}{8}$ to $\frac{1}{3}$ grain. The only difficulty is found in the insertion of the needle into the vein. Sometimes a little blood escapes from the vein under the skin, forming a small but painless sugillation. The injection can be made alternately in each arm. Six to ten injections in the course of treatment, at most, being necessary, it is well to begin near the wrist, and later inject nearer the elbow. If the weaker solution is employed, daily injections are in order; if the stonger, then once in two days is sufficient. The mouth and teeth must be carefully looked after and cleansed. For this purpose potassium chlorate is used as a mouth-wash. Of the eleven patients under observation, one developed diarrhœal stools, with small amount of blood, and two albuminuria, but casts were not found. The results which were observed from this treatment were: 1. The pains were lessened, and consequently sleep was permitted. 2. Swelling was remarkably diminished in the joints primarily affected, but secondary infection of other joints was not prevented. This is not an argument against the use of this method, for it occurs with others—*e. g.*, the use of salicylates. 3. If the injections are given in the morning, the expected afternoon rise of temperature is postponed for several hours, lessened, or abolished altogether. The indications for this method are: Acute articular rheumatism of infectious initial processes (erysipelas, phlegmon, puerperal affections) or peculiar history (chills, recurring endocarditis, frequent exacerbation, protracted subacute course, metastases) which point toward a pyæmic character. In acute disease, when the salicylates are forbidden, or when the inflammation is localized in single joints, this method is recommended. The contraindications are to be found in very weak individuals, severe renal degeneration, and by onset of severe symptoms of poisoning.—*Centralblatt für die Gesammte Therapie*, 1898, 1 Heft, S. 1.

Globularine and Globularetine.—Dr. Poucel reports that in the forms of arthritis—rheumatism, gout, gravel, the cutaneous or mucous manifestations, the auto-intoxications, by retention of nitrogenous metabolism producing acceleration or irregularities of the heart, extractive matters producing hyperpyrexia, toxins paralyzing the myocardium—these remedies combined produce a cure or relieve the symptoms with surprising rapidity. Brief histories of eight from the two hundred instances of its use are reported. The dose is about a grain of the former and rather more of the latter substance. If administered as the prasoïde of Heckel the dose is from sixty to ninety drops per day. Clinically, this remedy is a visceral depurative and a cardio-vascular tonic.—*Bulletin Général de Therapeutique*, 1898, 3e liv., p. 81.

[Globularine is a bitter glucoside found in different species of globularia. Globularetine is a decomposition product of this. These are probably obtained from the Globularia alypum, the wild senna of Europe, which grows along the shores of the Mediterranean.—R. W. W.]

The Sterilization of Urethral Instruments with Paraform.—Dr. Edward Martin makes use of a box of wood or metal, or of any comparatively air-tight material, in which the instruments can be laid, with a tray or cage upon which the paraform can be spread so that the vaporization may be fairly rapid. In his experiments thirty grains of the substance were used. For efficient lubricants he makes use of two preparations: (1) purified liquid petrolatum with a small quantity of menthol or oil of cinnamon; or (2) a 33 per cent. boroglycerin preparation.—*The Philadelphia Polyclinic*, 1898, No. 6, p. 60.

Methylene-blue in the Treatment of Acute Gonorrhœa.—Dr. Orville Horwitz has employed the following formula in one hundred and five instances: Methylene-blue, 2; oil of sandalwood, 3; oleo-resin of copaiba, 3 grs.; oil of cinnamon, 1 drop; given in capsules. When this combination is administered the purulent discharge, together with all inflammatory symptoms, usually disappeared within four or five days. He presents the following conclusions: 1. Methylene-blue is a germicide of great value in cases of acute urethritis due to the presence of gonococci. 2. It will not abort the disease, but will materially shorten its duration. 3. It markedly lessens the tendency to complications. 4. It is not to be employed in acute urethritis unless gonococci are present. 5. It should be employed as early as possible. 6. Its action is enhanced and the duration of the disease shortened by combination with copaiba, sandalwood, and salol. 7. Injections and irrigations of potassium permanganate early, and, later, astringent injections have a marked tendency to lessen the duration of the disease. 8. It colors the urine a deep-blue; of this the patient should be informed. 9. It is of no service in non-specific urethritis.—*The Philadelphia Polyclinic*, 1898, No. 8, p. 98.

The Treatment of Syphilis.—Dr. Richard Kopper presents a plea for the hypodermatic use of corrosive sublimate in large doses. He makes use of the following formula: corrosive sublimate, 5; sodium chloride, 5; distilled water, 100; and reports the results of 258 injections in 69 hospital

patients, and 240 injections in 58 ambulant patients. The injections are made into the gluteal region a short distance from the anus and avoiding a locality which will interfere with sitting or locomotion. The injections are to be placed deep into the muscles (one and one-half inches), and the entire quantity deposited before withdrawal of the needle, so that none of the solution will get into the track of the needle, because this is painful. The orifice should be closed with iodoform-collodion. Intra-muscular infiltration occurs in about one-fifth of the cases. This may become the size of a bean or a walnut, but rapidly disappears. Abscesses have not occurred. The interval between injections should be from five to eight days.—*Prager medicinische Wochenschrift*, 1898, No. 1, S. 1.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

PROFESSOR OF MEDICINE IN THE JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND,

AND

GEORGE DOCK, M.D.,

PROFESSOR OF MEDICINE IN THE UNIVERSITY OF MICHIGAN.

Disturbance of the Choking-reflex, Speech, and Deglutition in Hemiplegia.—W. KATTWINKEL (*Deutsches Archiv für klin. Med.*, Bd. lix. p. 317) has made an investigation under Prof. P. Marie, at the Bicêtre, Paris, for the purpose of finding some anatomical ground for the symptoms named, so important in diseases so various as pseudobulbar paralysis, hemorrhage, infantile cerebral palsy, etc. He examined especially one hundred cases of hemiplegia, fifty right-sided and fifty left-sided. The age of the hemiplegia, its origin, whether apoplectic or gradual, and the extent and duration of disturbances of the functions mentioned above were investigated. The methods of clinical examination require no description. The principal conclusions drawn are: 1. That the centre for word-forming is in Broca's convolution. 2. The centre for co-ordination of speech—articulation—is chiefly in the third right frontal convolution. 3. Both centres are united by association-fibres passing through the corpora striata. 4. The reflex centre for the pharynx and larynx is in the corpus striatum, especially in the right side, where also the deglutition centre lies.

The author found disturbances of speech in forty-one out of fifty cases of left-sided hemiplegia, and in twenty-five of these the disturbance was permanent. They consisted essentially of loss of co-ordination, and are ascribed, in opposition to most writers, to the lesion in the third right frontal convolution, and not to functional alterations of the facial or hypoglossal nerve. A number of the cases are cited, some with autopsies.

Thrombosis of the Portal Vein.—This is an extremely rare condition, but one that can at times be recognized during life. BORRMANN (*Deutsches*

Archiv für klin. Med., Bd. lix. p. 283) has worked up the previously-reported cases and added one of his own. He shows that besides thrombi of the portal vein, due to compression or to the spread of inflammation from the vicinity, there are others, which may be called primary, depending on a local disease—atheroma—and having an etiological relationship, among other things, with syphilis. The clinical picture of this process is often not striking, but may be very characteristic. In the latter case there is a sudden onset with pain and all the signs of an acute stasis in the portal circulation—ascites, vomiting of blood, bloody stools, enlarged spleen. Of diagnostic importance are the condition of the liver, the history, and the suddenness and severity of the symptoms. If there is a complete and sudden obstruction of the portal vein, death ensues; but in other cases canalization of the thrombus and temporary restoration of the lumen permit the patient to live a long time. The consequences of thrombus for the liver are not so important as might be supposed, and consist chiefly of moderate atrophy and interstitial sclerosis. An important sign is rapid recurrence of ascites after puncture.

A New Method in the Radioscopy of the Stomach and Intestines.—I. BOAS and M. LEVY-DORN recommend (*Deutsche med. Wochenschrift*, 1898, No. 2) an ingenious method. Gelatine capsules are filled with metallic bismuth and then covered with celluloid, making them insoluble. To aid recovery from the stools the capsules are colored with a non-poisonous aniline dye. The capsules are 2½ cm. long, 1¼ cm. wide, and weigh when full twelve grammes. They are readily swallowed, and in none of the fourteen persons experimented on were any symptoms produced. The capsules are readily seen (in thin persons) with the radioscope; the position in the abdomen may thus be recognized, but the exact part of the alimentary tract can, of course, be determined only within certain limits. Practically, it was not difficult to know when the capsule was in the fundus of the stomach, or in the cæcum, because it remained there longer than in other positions. In the small intestine the progress seemed to be rapid, and when the capsule was found in the left iliac fossa it was evacuated within twenty-four hours, indicating its presence when seen before in the sigmoid flexure.

The capsule usually disappeared from the stomach within twenty-four hours in patients with mild gastric symptoms, and was then found in the cæcum; but in obstruction of the pylorus it remained as long as four or five days in the stomach.

The possibilities of this method of examination are evidently great. The motion of the intestinal tract in health and disease, and under the influence of various drugs, especially those like creosote, alcohol, strychnine, bitters, and cathartics, can be studied with advantage, though, as the authors remark, conclusions as to the motion of the usual ingesta must be drawn with caution.

A Case of Chronic Fibrinous Bronchitis; and the Nature of the Casts in that Disease.—A. HABEL reports a case from Eichhorst's clinic. A woman, aged forty-one years, with long-standing mitral insufficiency and stenosis, expectorated fibrinous casts almost daily for about two weeks. Later

she had an incarcerated hernia, was operated upon and recovered, but developed nephritis and a recurrence of the fibrinous bronchitis. Finally, loss of compensation came on, and the patient died without further fibrinous expectoration. Habel made examinations of the casts in this and another case, also secondary to valvular disease. Stained by Weigert's method, they showed very few fibrin fibres, most of the casts not taking any stain (lithium carmine). Chemical examination also showed the absence of fibrin, but proved that the casts were made up chiefly of mucin. The casts were of acid reaction, and the author thinks this is the cause of the coagulation. According to his view something, probably the action of bacteria, causes the bronchial secretions to become acid. The mucus then coagulates. He applies the same explanation to the casts sometimes expectorated in croupous pneumonia, and was able to confirm his view in a case of the latter disease. —*Centralblatt für inn. Med.*, 1898, No. 1.

The Serum-test for Typhoid Fever at the Montreal Meeting.—The important papers read go to prove that, however great the scientific interest of the serum-test, its results must be used with caution in practice. WIDAL showed, from the extensive statistics of CABOT, that the reaction is present in a very large proportion of cases. He himself only missed it once in 177 cases. He emphasized the difficulty of diagnosticating clinically all cases of typhoid fever. In doubtful cases he had made cultures from the spleen. By this method he was able to demonstrate the absence of reaction in a case of genuine typhoid fever with relapse. The examinations were negative during both febrile periods, the intervening stage and the convalescence. Such cases are so rare as hardly to affect the value of serum diagnosis, but prove that the agglutinative reaction has nothing to do with immunity. They follow naturally after observations showing that in some cases the reaction comes on very late. Widal believes the reaction is often more intense in the beginning of the attack. It diminishes and frequently disappears from the first period of convalescence, and is relatively rare in patients who have been cured for more than a year. W. GILMAN THOMPSON described observations showing that the reaction often became more intense as the disease progressed. WILSON and WESBROOK related an interesting case in which there was reaction on the twelfth, nineteenth, twentieth, and twenty-fifth days; absent on the ninth, thirteenth, and twenty-first days, without corresponding variation in the clinical symptoms. These observers examined a large number of cases in children, and found a reaction in 76 out of 165. The reaction appeared earlier than with older cases. (The clinical features in these cases are not mentioned.) In one case a mother seven months pregnant had clinical symptoms of typhoid, though her blood failed to react on the fourth and sixth days. The child was born on the sixth day, and two days later gave a marked reaction. The mother reacted on the thirteenth and twentieth days. E. BATES BLOCK gave some interesting and valuable details on the technique, and advocated the use of serum, rather than dried blood. Thompson held that the value of the test was about the same as that of the diazo-reaction, "confirmatory in connection with appropriate symptoms, but misleading if positive reliance be placed upon it;" but MUSSER and SWAN seemed to have had a more fortunate experience. The

latter observers called attention to the fact that the presence of the reaction does not lessen the importance of making an accurate diagnosis of the patient's condition as a whole, illustrating this by the history of a patient who might have been thought to have a relapse, but proved to have a meningitis following the typhoid attack.—*British Medical Journal*, December 18, 1897.

Hæmatoporphyrinuria, with the Report of a Case.—OGDEN (*Boston Medical and Surgical Journal*, February 24, 1898, p. 169) gives a concise historical review of the subject of hæmatoporphyrinuria, and reports a personal observation.

Hæmatoporphyrin is a rare constituent of the urine, and is a coloring matter derived from the blood. The first observations on the subject were made by Hoppe-Seyler in 1871, when he found that hæmatin heated with sulphuric acid yielded a compound whose acid and alkaline solutions showed spectral bands unlike any substance he had seen before. To this new compound he gave the name "hæmatoporphyrin." Nencki and Sieler determined its exact formula to be $C_{16}H_{18}N_2O_3$, and the former demonstrated that the only chemical difference between hæmatin and hæmatoporphyrin was that the latter was simply hæmatin free from iron.

In 1880 MacMunn described a pigment which he obtained from the urine of a case of acute rheumatism, and which he called "urohematin." In 1883 he reported other cases of acute rheumatism in which this pigment occurred, and from the similarity of its spectrum to that of hæmatoporphyrin he named the pigment "urohæmatoporphyrin."

Hæmatoporphyrin has been found in the urine in pulmonary tuberculosis, in pleurisy with effusion, acute rheumatism, lead-poisoning, and intestinal hemorrhages. The pigment has been found probably most frequently in the urine of patients to whom sulphonal had been administered. In fact, it seems to occur almost constantly when the drug has been given in considerable quantities, but never when only a single dose (one gramme) has been taken. It has also been found in the urine after the administration of trional and tetronal.

Stokvis has found that it occurs after intestinal hemorrhage. In artificial hæmatoporphyrinuria produced in rabbits by the administration of sulphonal, he found that minute hemorrhages into the mucous membrane of the stomach and intestines occurred. From these areas he extracted hæmatoporphyrin, and he believes that in these cases the blood which had entered the digestive tract was changed into hæmatoporphyrinuria, absorbed, and excreted in the urine.

Ogden's case occurred in a woman, aged thirty-eight years, who had suffered from rheumatism for years, and who had a mitral heart lesion. The patient previous to the appearance of the hæmatoporphyrinuria had had diphtheria. During convalescence she began to have vomiting and diarrhœa, and later pains and loss of sensation in the legs and body up to the waist line. Urine and feces were passed involuntarily. On two different occasions trional in fifteen-grain doses was given for insomnia. It was after the administration of this drug that the dark, pigmented urine containing hæmatoporphyrin was voided, and Ogden believes that the drug was responsible for its

occurrence, although he admits the possibility of the disease of the nervous system and the gastro-intestinal disturbance being the causes.

Chronic Diphtheria.—C. MEYER, in a valuable report on the modification of diphtheria during treatment with antitoxin, records two interesting cases (*Deutsches Archiv für klin. Med.*, Bd. lix. p. 465). The first, a boy two years old, was ill a week before admission. Two days before the latter there was hoarseness, and one day dyspnœa and epigastric retraction. One thousand antitoxin units were injected. On admission there was an extensive grayish-white membrane in the pharynx; the submaxillary lymph glands were enlarged; there were cyanosis, laryngeal stridor, hoarseness, and frequent cough; pulse and respiration accelerated; temperature normal. There was also bronchitis, but no pneumonia. The membrane gave an almost pure culture of diphtheria bacilli. A second injection of 1000 units was made at once, and a third the next day but one. After that the stridor disappeared, but, as the respirations were more frequent, cough worse, and temperature higher, another injection was made. This was followed by a macular eruption. Respiration was still frequent; there were moist râles and cyanosis. Four days later the temperature was 102.5°, and, on account of the progressing disease in the bronchi, another injection was made, followed again by a partly hemorrhagic, partly macular eruption, and a diffuse œdema of the arms and legs. The fever continued another week, when desquamation occurred in large scales. Twenty-five days after admission tracheotomy was performed on account of the marked stridor and the weak respiration over both lungs. Pus from the trachea contained diphtheria bacilli in almost pure culture. A week later the patient died. Autopsy disclosed diphtheritic and streptococcus infections of the lung, in small foci, necrotic in the centres. The author considers the long duration of the disease as an evidence of the specific action of the antitoxin. The passive immunity served to prolong life, but notwithstanding, or, perhaps, because of it, the body could not furnish active immunity for definite recovery. Perhaps also the incidental effects of the remedy in weakening the body contributed to the fatal end, though not the chief cause of death. In the second case, a three-year-old girl had laryngeal stenosis and left-sided pneumonia. Cultures showed diphtheria bacilli. During the first six days two injections of 1500 units each of Behring's serum, and one of 1000 units, were made. Dyspnœa growing worse, tracheotomy was performed. On the following day an eruption appeared, with high fever, lasting nine days. The canula was then removed; the pulmonary symptoms improved, but fever continued. Membranes were twice expelled. For more than a month bronchitis continued. Frequent cultures always contained Loeffler's bacillus. Three months after the beginning tracheotomy was again necessary, and was followed by bronchitis and pneumonia. An injection of 1000 units was followed by an urticarial rash. Cough and hoarseness were noted occasionally after this at intervals. The bacilli were found in the secretions up to a year and a half after the primary attack, the pharynx showing no abnormality.

Scarlet Fever.—C. SEITZ publishes some interesting data and conclusions, based on observations extending over ten years (*Münch. med. Wochenschrift*,

1898, No. 3). He was unable to find either bacteria or protozoa as primary causes, and looks on streptococci as secondary invaders. The variability of the death-rate, so often mentioned, was very striking, as shown by the extremes of mortality in different years: 2.6 and 15 per cent. Season and weather had no definite influence on the frequency of complications. The local infectiousness of certain houses and rooms, and the family disposition, were often observed, as also the temporary individual disposition, many persons being affected only after several weeks' exposure. As to age, the first year of life showed the smallest morbidity, but the greatest mortality—40.6 per cent. Most cases occurred between the second and fifth years. Out of 833 cases second attacks were twice noted, once after one and once after two years. In the latter the second attack was the more severe. Recidives of the rash were often noted, usually after eight to ten days. Nephritis was not affected by season, nor by the diet, but was dependent on the character of the reigning epidemic. Although the patients lived under practically uniform conditions, the frequency of nephritis varied from 9 to 41 per cent. in different seasons. The greatest proportion happened in the summer, and the complication, sometimes hemorrhagic, sometimes with uræmia, occurred even in children on milk diet and in bed. The author, of course, does not advise neglect of these features. He believes that many cases of so-called intermittent albuminuria and chronic nephritis are due to unrecognized or neglected scarlatinal nephritis. Seitz thinks the angina of scarlet fever is rarely diphtheritic. Out of 800 cases 200 had pharyngeal false membranes, but paralysis followed in only one case. Of 98 cases in which cultures were made, Loeffler's bacillus was numerous in three and scanty in four, in repated cultures. He, therefore, rejects antitoxin in scarlatinous angina and uses Heubner's injections of 3 per cent. carbolic acid, in the tonsil and soft palate, with cleansing and antiseptic applications by gargle, spray, and swab.

Myoma of the Stomach.—HERHOLD reports an example in a woman of thirty-seven years, with a history of intractable vomiting soon after eating, for three years. Palpation of the abdomen was negative. The stomach was moderately dilated. The vomitus contained free hydrochloric acid, and, although vomiting always occurred soon after meals, not everything eaten was vomited. As there was a history pointing to old peritonitis, it was supposed there was an adhesion pulling on the pylorus. Dietetic and medicinal treatment having failed, laparotomy was performed. In the pyloric region a tumor the size of a hazel-nut was found obstructing the passage into the duodenum to a moderate degree. The tumor proved to be a myoma. Recovery was complete. The author explains the vomiting as due to a reflex, brought on in the neurasthenic patient when the food reached the pyloric region. He could find only two other cases reported in the last ten years. In both the tumors were much larger than in the one now reported, in the case of von Esbach weighing five and one-half kilos.—*Deutsche med. Wochenschrift*, 1898, No. 4.

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

PROFESSOR OF CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE UNIVERSITY AND PHILADELPHIA HOSPITALS;

ASSISTED BY

ALFRED C. WOOD, M.D., INSTRUCTOR IN CLINICAL SURGERY, UNIVERSITY OF PENNSYLVANIA; ASSISTANT SURGEON, UNIVERSITY HOSPITAL.

AND

C. L. LEONARD, M.D., ASSISTANT INSTRUCTOR IN CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA.

The Food and Digestion after Complete Removal of the Stomach—Œsophagoenterostomy.—The complete removal of the stomach in man is an operation seldom attempted and more rarely successful as regards the future of the patient. SCHLATTER (*Correspondenzblatt für Schw. Aerzte*, December 1, 1897) reports the following interesting successful operation:

The patient, a female, was fifty-six years of age. She had always had trouble with her stomach from childhood, and, of late, pains and vomiting. Blood and bile were occasionally present. Medicinal treatment was of no avail, and, a tumor being recognized, she was sent to the hospital. She was very thin. Palpation showed a tumor, oval in shape and the size of two fists, occupying the region of the stomach. She was unable to retain any food except milk, and desired operation. There was no free hydrochloric acid.

The exploratory laparotomy showed a hard tumor extending from the pylorus to the cardiac end of the stomach; there were at the pyloric end three soft glands, and the tumor was mobile. Gastroenterostomy was impossible, as the whole stomach was invaded, and the only alternative was the total removal of the stomach.

The stomach was drawn down, the œsophagus seized with a Wölfler's compression-forceps, and the œsophageal end of the stomach with a Stille's forceps, and then resected. Access was obtained to the œsophagus by tearing through the greater and less omentum with Pean's forceps, and ligating with silk. The resection at the pyloric end was done in a like manner. The lymphatic glands were readily removed by blunt dissection. It was found to be impossible to connect the duodenum and œsophagus end to end. The duodenal end was therefore tucked in and closed by a double row of sutures. The duodeno-jejunal fold was then sought, and, when found, it was readily connected to the œsophagus by a lateral and end anastomosis by a series of interrupted silk sutures in the mucosa, and of Lembert sutures in the serous surfaces. The release of the ends allowed the œsophagus to retract, so that the line of union was brought up toward the diaphragmatic œsophageal opening. The abdominal wound was then closed and a dressing applied. A pathological and histological examination of the ends of the tumor showed that the entire stomach had been removed and that there were portions of the œsophagus and duodenum on the tumor-mass removed.

Nutritive enemata were given containing eggs, milk, and brandy. The patient's pulse after operation was 96 and evening temperature 36.4° C. The following day the pulse went up to 142, but the temperature was 37.3° C. The enemata were continued, and during the afternoon tea, with milk, was given per os, and was well borne. The second day the enemata were not retained, and a half glass of Bordeaux was given, a teaspoonful at a time; the temperature rose to 38.1° C. and the pulse to 160, but remained full and strong. The patient had sharp attacks of pain in the region of the stomach. The third day milk, eggs, bouillon, and wine were taken in small doses every two hours, with pepsin and dilute muriatic acid. The pulse was 146 and the highest temperature 38.1° C. On the seventh day the dressings were removed and the wound found to have healed per primam. The first stool was on the fourth day; thereafter, two to three fluid stools. Occasionally there was some milk regurgitated. Ten days after the operation the patient vomited; the diet had become much greater in amount, though administered in small doses.

Twenty days after the operation the patient had for dinner half a chicken, and rested till 4.30 P.M.; she took her usual milk and egg at 6.30; at 7.30 she vomited, with marked contraction of the abdominal muscles, together with considerable choking. There were about 300 c.cm. of fluid, containing the milk, some egg taken an hour before, and some shreds of flesh. After vomiting the patient had a bitter, gally taste in her mouth. On several other occasions the patient vomited. Chemical examination showed the absence of free hydrochloric acid and the presence of lactic acid, with the evidence of the action of trypsin upon the food. Gallic acid and bile pigments were also present. The pepsin and hydrochloric acid were discontinued.

In the first two months after the operation the patient gained about 4400 grammes.

The relation of the stomach to digestion and the bearing upon nutrition, after operation of the total removal of the stomach, the author believes have been shown by this case to be somewhat different from the physiological function usually ascribed to it.

In these cases the disease has so modified the capacity of the stomach and its secretions that it is far from normal, and the system is, as it were, accustomed to doing without it before it is removed. The study of the necessary modification in the diet which the operation required showed the author what the physiological function of the normal stomach was and what loss the system had sustained.

His investigation showed that one of the chief functions of the stomach is that of a reservoir. The food is retained here for a greater or less length of time, according to its digestibility, preventing the intestines from overloading. Extremes of temperature are also corrected, and their harmful action on the intestines prevented. Food was given in small amounts, frequently repeated, and of a character most easily assimilated.

The chemical action of the stomach was at first supplied by pepsin and hydrochloric acid, but the pepsin was useless in the alkaline fluids of the intestine, and the hydrochloric acid was neutralized, so both were discontinued. The action of the stomach on bacteria and in restraining decomposition was

found to be very slight; at least their absence produced no marked change; while the absorption attributed to the stomach is of a very slight degree.

Observations on the relative rapidity of the passage of food through the alimentary tract did not show any marked acceleration, and the chemical analysis of the urine showed no marked alteration.

In this patient the author studied carefully the mechanism of vomiting, and defines it as a complicated act with a number of movements of the muscles of the stomach, œsophagus, the muscles of swallowing, the diaphragm and abdominal muscles. The complicated movements can only arise from a centric source, and these vomiting centres may be stimulated reflexly in a variety of ways.

The gain in weight which the patient showed, which persisted for so long a time after the operation, shows that the stomach is chiefly a protective organ for the intestines and that the intestines, with proper feeding, are capable of entirely replacing the chemical action of the stomach.

On the Immediate Reduction of the Deformity of Pott's Disease of the Spine.—TUBBY (*The Practitioner*, January, 1898), who has performed this operation sixty times, says that it is applicable only to certain cases, and that the following may be regarded as entirely unsuitable: 1. Those cases in which tubercle exists elsewhere. 2. Cases in which much wasting is present there; the pressure of the splint or the plaster-of-Paris jacket, which is used afterward, is apt to give rise to sores. 3. Children who suffer from a cough or other respiratory trouble. 4. Cases in which abscesses are present. 5. Cases in which firm ankylosis has taken place. 6. Cases in which considerable alterations in the shape of the bony framework of the chest have occurred. 7. Patients over twenty years of age are, as a rule, not suitable subjects. 8. Cervical curves are not suitable, for obvious reasons, and in cervico-dorsal curves it is difficult to maintain the reduction on account of the leverage arising from the weight of the head. The size of the curve, provided it is recent, is not necessarily a hinderance, since experience has shown that curves of moderate size yield more readily than small projections.

The cases which are suitable are those in which the disease is recent; in which the angle of curvature is a changing one, the patient is under twenty, the general health fair, and the disease is not so active as to give rise to general constitutional disturbance. Lower dorsal and lumbar curves are, *cæteris paribus*, the most suitable for reduction. *A priori*, the method would seem certain to produce paralysis, but cases that were paretic and paraplegic recovered complete movement and sensation subsequently to the operation.

The author then gives in detail the method of preparing the patient and of performing the operation. He prefers to use a modified double Thomas' hip-splint, with head-rest, rather than the plaster-of-Paris dressing, as it is possible to get at the patient more readily if there is any respiratory complication, and there is not the same danger of producing sores. The patient must be carefully watched out of the anæsthetic. After a few days in the hospital the patient can be sent home, to return once a fortnight for inspection. At the end of two months the splint is removed to ascertain the condition of the parts.

The author does not report the ultimate results in any of the cases treated,

but says: "It appears that in suitable cases this method opens out a good prospect of cure, with a straight spine. And, inasmuch as the method can be carried out with safety, it seems to have a distinct future before it. But the risk is that it may be attempted upon unsuitable cases, and in such an event it can only be that discredit will be thrown upon the procedure."

Contribution to the Surgery of the Bladder.—After remarking on the fact that neoplasms of the bladder exhibit the peculiarity that whether they be simple or malignant they all prove eventually fatal, unless prevented by surgical intervention, CAIRD (*Scottish Medical and Surgical Journal*, December, 1897) calls attention to the progress which has been made in the operative treatment of these tumors. Perineal drainage was a great step in advance in relieving these conditions and checking hemorrhage, and is to-day one of the safest palliative measures in the majority of deeply-infiltrated tumors. The safety of the high or suprapubic operation has been greatly increased by antiseptic measures and improved methods, while the relief to the patient is as great and the inconvenience less, as, by the latter route, the urine can be retained by a proper device, so that the patient can get about.

The only other alternative is complete removal of the growth in malignant cases, and the cystoscope is the means by which we can decide beforehand whether operation can be undertaken or not, and how extensive it must be.

The amount of involvement and location of the tumor have much to do with the prognosis: does it involve the ureters, one or both? is it extraperitoneal or not? These considerations affect the gravity of the operation. If the tumor can be entirely removed without section of the ureter, the prognosis is less grave, while section of one ureter is less grave than of both. They must necessarily be replanted; then where? In the bladder, if possible; if not, in the rectum. Cases have been reported where such a procedure produced good results.

Traumatic Aneurism of the Ulnar Artery in the Palm Cured by Tying the Ulnar Artery Above the Wrist.—ROBERTSON (*British Medical Journal*, December 4, 1897) reports the case of a fireman who complained of a painful spot just under the hypothenar eminence of the left hand. On comparing the two hands one could plainly detect a bulging at the affected part, and with this bulging a pulsation was very noticeable. He had more than once remarked on the beating. It was, however, the pain that worried him. The swelling was nearly an inch in diameter and almost circular. Firm palpation increased the pain. The patient was healthy, had no syphilitic history, and had never had a day's illness. The traumatic origin was clear, the patient having five months previously given the injector of his engine a firm knock with this hand. This caused a sharp pain in the hypothenar eminence. The swelling and pulsation were first noticed two months ago.

Ligature of the left ulnar completely stopped the beating, the wound healed per primam, and no pulsation has returned. The author believes the extirpation of the aneurism a better operation. Its advantage lies in the fact that the future of such cases need give no anxiety.

Successful Removal of an Enormous Mesenteric Tumor and Nearly Eight Feet of Intestines.—SHEPHERD (*Montreal Medical Journal*, December, 1897) reports the removal of a fibromyxoma weighing thirteen pounds, and with it seven feet and eight inches of intestine. The tumor was one of gradual development and filled the entire abdomen. The operation showed it to be freely movable above, but attached by strong adhesions below. After removing the adhesions and delivering the tumor, it was found to arise from the mesentery and to have attached to it three feet of intestine. It was impossible to enucleate it, and after the mesenteric vessels had been tied the operator found that he must remove more intestine, as its blood-supply had been cut off. The ends of the gut were united by suture, and the patient made a good recovery, although it was necessary to inject salt solution while he was still on the table. The patient gained weight steadily after the operation, and six months later was presented to the medical society in perfect health, having gained twenty-five pounds.

Operation in Intestinal Obstruction.—THOMSON (*Dublin Medical Journal*, December, 1897) says of the typical symptoms of obstruction, pain, vomiting, and constipation, that they do not always form an infallible guide in these cases, and that other elements must enter into our consideration before we can form a definite diagnosis. The surgeon is not usually consulted early enough, not that operation would have perhaps been indicated earlier, for the surgeon is usually as conservative as the practitioner, but that, if operation is necessary, he who becomes the responsible actor should be able to select the most suitable time for his operation. The advisability of operating in every case is not agreed upon by surgeons, but the author's idea of the modern surgical view on this subject is, "because we are in the dark, let us let in the light."

Of the symptoms, he says of vomiting, it is especially valuable, but the quality of the vomit must be considered and the length of time it has continued. Persistent stercoraceous vomiting is an almost positive indication for operation. Pain is of value in locating the trouble within the abdomen, but it rarely indicates the actual site of the obstruction. It has, however, characters of its own. In chronic obstruction it is not severe, and it differs from that of the acute affection, which comes on suddenly and is usually intense. Sometimes there are intervals of quiescence; sometimes the pain may be almost continuous, and the difference seems to depend upon the tightness of a constriction and on whether there still remains any portion of the lumen through which matter may pass.

Temperature is often misleading, and is only to be mentioned to warn against reliance being placed upon it. On the constipation itself we must also not place too much reliance. It depends very much upon the position of the stoppage. If it is high up in the intestine motions may persist for some time, and even diarrhœa may appear for a day or two. If the stoppage is in the large intestine we have the constipation from the outset, although sometimes a loaded rectum below the point may puzzle us by discharging its contents.

Dulness in the flanks is a very unreliable sign. The presence of indican in the urine, and the suppression in proportion to the nearness of the stop-

page to the beginning of the small intestine, are localizing symptoms that are of value.

The general condition of the patient must, however, be considered: the facial expression, and the collapse in some form, which is always present and is sometimes progressive.

These are the series of symptoms, but they are not all present in every case, and skill in diagnosis depends on being able to select the symptoms present and to combine them so that they will lead us aright.

In the vast majority of cases of acute obstruction, to which the author confines himself, there is no room for medical treatment. It is only time lost. Taxis has no attraction whatever for the author. There is no certainty in location of the point affected or of telling how great the destruction of tissue is or what damage is done by the reduction.

We do not conduct the ordinary affairs of life upon the supposition that a miracle is likely to happen; and he supposes that a practitioner who relied on medicine to cure, say a strangulation, would be horrified if it were put to him that that is practically what he is doing. Granted the recognition of the character of the case, there is only one thing to be done, and that is to explore. It is not the opening of the abdomen that kills; it is the fact that that is usually done when, by delay, everything is arrayed against success.

Skiagraphy in Surgery.—In reviewing the progress made in surgery, CARLESS (*The Practitioner*, January, 1898) says of skiagraphy that "fractures are constantly being submitted to examination by its means, and one effect in this direction will be that the general public, which is now able to ascertain without difficulty or much expense whether or not such lesions have been satisfactorily treated, will demand a much more exact correction of the resulting deformities. The patient has the right to demand from the practitioner a perfect restoration of the *status quo ante*, or to know why such cannot be obtained." The author agrees with the suggestion that fractures should be called open and closed rather than compound and simple, and says: "Many a so-called simple fracture is, in reality, much more serious than one called compound, and the patient who recovers from such a lesion may well be dissatisfied with a result which leaves the limb deformed and with its utility considerably limited. One outcome of skiagraphy will certainly be a more frequent resort to operative proceedings." Since the fear of sepsis has been banished there is really no reason why, in careful hands, closed fractures should not be laid open; the risk associated therewith is minimal, the advantage to be gained incalculable. This particularly applies to oblique fractures, whether of the arm or leg, where it is difficult to command the fragments with splints. By this means blood-clot, which, when organized, forms such an obstacle to satisfactory healing, can be removed, while the fragments are wired or pegged together in proper position.

A Case of Typhoidal Cholecystitis with Cholelithiasis.—The case reported by MARTIN (*Montreal Medical Journal*, December, 1897) had the following symptoms: Female, aged thirty-five years; had a temperature of 98⅘°, pulse 90, respirations 24 per minute. She was poorly nourished, evi-

dently suffered intermittent pain; had grayish-coated tongue, anorexia, and a diarrhœa, with clay-colored stools. There was no jaundice. Examination of the abdomen revealed slight redness over the right hypochondrium and a scarcely perceptible œdema. Pressure on the ribs induced tenderness, but no friction-rub could be felt on palpation. Immediately below the ribs a large mass was detected, continuous with the liver and extending down for as far as 2 to 3 cm. below the umbilicus, situated toward the median line, and its right border slightly external to the right rectus muscle. Percussion over this area produced a modified dulness, with some slight tympany. From above downward the hepatic dulness began at the sixth rib. Pressure behind gave slight tenderness in the right line, but elsewhere there was no evidence of disease. The urine showed a trace of albumin, but no bile. The blood examined in two different laboratories gave the Pfeiffer test for typhoid. The differential diagnosis lay between subcutaneous phlegmon, appendicitis, and infection of the bile-passages as the conditions which could not be readily excluded. The œdema pointed to cellulitis, while some more deeply-seated condition was also probably present. The mass itself could not be definitely defined, except what seemed a tongue coming down from the liver. There was no distinct fluctuation. The symptoms, with the clinical history, pointed rather to some infection of the gall-bladder than to appendicitis. The ultimate diagnosis was that of cholecystitis and cholelithiasis, and operation was urged.

The operation substantiated the diagnosis: a distended gall-bladder was found, from which 150 small facetted stones were removed and a large amount of turbid, grayish-green fluid. The patient did not recover, and died at the end of the second day from the disease. A partial autopsy showed the characteristic lesions of typhoid fever in the intestines and the subacute inflammation of the gall-bladder itself, with the more or less subacute pericholecystitis.

Cultures were obtained at the operation from the gall-bladder, and microorganisms obtained which responded to all the tests for the B. typhosus.

The sudden alteration in the course of the disease, with development of a subnormal temperature and sudden pain in the abdomen, might have suggested perforation of the intestine; but, the pulse remaining quiet and strong, this was practically excluded. The symptoms, too, were intermittent, with periods of comparative freedom from pain and anxiety, which, too, has been a feature prominent in many similar cases of typhoidal cholecystitis. Add to this the tongue of liver pulled down by the gall-bladder, and we have a series of suggestive symptoms for the diagnosis. Jaundice was, of course, not to be expected, as the other bile-passages were free. The condition found shows the necessity for immediate operation in such cases.

Nephrectomy and Nephrolithotomy.—In connection with some highly interesting cases, MYLES (*Edinburgh Medical Journal*, September, 1897) discusses the regional anatomy of the kidneys and shows that the practice of needling is fraught with grave danger. He summarizes his views as follows, giving his objections as:

1. The limited space available for preliminary introduction of the needle into the kidney.

2. The danger of wounding the hollow viscera and inoculating the kidney from them.

3. The danger of wounding the renal vessels, or even the cava.

4. The necessity for many punctures to insure thorough exploration.

5. The difficulty of determining to what depth the needle can be safely thrust.

6. The danger of local necrosis of the kidney.

Of the methods of operating upon the kidney he says there are only two methods worthy of consideration: The intra-abdominal route, the lumbar incision and the conjoint method. He prefers the lumbar incision, for the following reasons:

1. It undoubtedly offers the easiest and safest route to the kidney.

2. Through it the pedicle can be easily secured before any attempt to detach the kidney from its bed.

3. Through it the kidney can be more easily and safely separated from adherent neighboring organs.

4. If the kidney is the seat of a large abscess, such abscess can be readily and safely drained without danger of peritoneal infection, before any attempt is made to deliver the kidney through the wound.

5. In case a calculus should be impacted in the ureter, unless it be below the pelvic brim, it can be more easily dislodged from the loin than from an abdominal opening.

6. The danger of infection from the exposed end of the ureter is avoided by the lumbar incision.

The only valid objection against this method is that a very large kidney cannot be delivered through it.

Against the anterior route he urges the following:

1. The additional danger of peritoneal infection.

2. The difficulty of manipulating the pedicle through a narrow aperture, liable to be blocked at any moment by rebellious coils of intestine.

3. The great difficulty that exists in detaching the kidney, if adherent, at such depth from the surface.

4. The great liability of tearing the thin-walled renal vein in the necessary manipulations.

5. The possibility of wounding the vena cava.

Mixed Tumors of the Soft Palate.—After a study of the pathology, clinical characteristics, evolution, diagnosis, and treatment of these tumors, BERGER (*Rev. de Chir.*, July, 1897) summarizes his views as follows:

Mixed tumors of the soft palate form a well-defined group, having marked anatomical and clinical characteristics.

These tumors have their origin in the glandular structures of the soft palate; their limits are well marked, and remain so, by an envelope of connective tissue which enfolds and isolates them entirely from neighboring tissues.

They justify the name which has been applied to them of epitheliomata of varying structure. Their composition is the following:

(*a*) Epithelial elements of which the nature is sometimes similar to that of adenomata, sometimes and more often that of epitheliomata.

(*b*) A connective-tissue web in which are found the various forms of connective tissues, principally that of mucous and cartilaginous tissue. The cases observed by the author are contrary to the theory of an endothelial origin of these tumors.

From the clinical stand-point these tumors are essentially benign; they have never, in the author's experience, presented the progress and termination of true epithelioma. This benignity seems to be due to the strangulation of the epithelial tissue of the tumor by the mucous or cartilaginous structure of the framework.

These mixed tumors of the soft palate are nevertheless difficult to distinguish from sarcomata, which, in this region, may present analogous characteristics—slow development, a very well-marked encapsulement, and a relative benignity.

The gradual growth of these tumors is their sole source of danger, injuring the function of neighboring organs by the compression which they exercise. The prolongations which they send out into the pharynx and the pterygo-maxillary and parotid regions, render their enucleation more difficult. This, however, can be accomplished through the help of the capsule which surrounds them. Recurrences are always due to the incomplete removal.

Other tumors of the soft palate often occur which present a marked analogy to these mixed tumors. But these tumors perforate the palate and invade nasal fossæ and maxillary sinuses.

They are less sharply defined than these tumors of the soft palate. Though some of them apparently arise from the palatine glands, and present the same texture as the mixed tumors, there are others which belong to the sarcomata, particularly the plexiform sarcomata and the angio-sarcomata.

These tumors of the palatine arch must not be confounded with the tumors of the soft palate. Their anatomical study demands new research. The diagnosis of their various forms cannot be established in a precise manner. Their prognosis should be much more reserved than in the mixed tumors.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

J. SOLIS-COHEN, M.D.,

OF PHILADELPHIA.

Illumination.—Dr. L. Lichtwitz, of Bordeaux, extols, above all sources of illumination (*Annales des Maladies de l'Oreille, du Larynx*, etc., 1897), that from acetylene produced automatically, as required, from a generating apparatus. He states that it is so white that the incandescent electric light appears yellow by its side.

Nasal Hydrorrhœa.—Mr. Cresswell Baber, at the meeting of the Laryngological Society of London, January 12, 1898, reported a case of profuse nasal hydrorrhœa in a lady forty-two years of age, which seems to have been controlled by application of the constant electric current externally on the nose for five minutes twice a day for some three months. The applications were entrusted to the patient herself.

Intranasal Sarcoma.—Dr. James E. H. Nichols reports (*New York Medical Journal*, January 8, 1898) four cases, in three of which extensive external operations had been practised, and in all had failed to restrain the progress of the growths, though in one instance, a girl of seventeen, life was prolonged for several years.

[It has been the experience of the compiler, that extensive operations are very rarely successful, unless the growth is circumscribed, and that the patient will live longer without operation. It is almost, if not absolutely, impracticable to eradicate the remoter ramifications of the neoplasm; hence sometimes the tumor has grown more rapidly after the operation than it did before, there being no tissue to restrain rapid development.

Sarcomas in their early stages, when accessible to their outer ramifications, can usually be successfully removed without danger of recurrence. If, as often happens, the cases are mistaken for ordinary polyps or other benignant growths in their early stages, the only chance of cure by radical extirpation is lost, and palliative and systemic treatment is the only alternative.]

Intranasal Angioma.—Dr. Glasgow (*New York Medical Journal*, January 8, 1898) reports a case of angioma in the nasal passage of a lady, twenty-two years of age. The diagnosis was assured by the microscopic examination, which was illustrated. The tumor was eradicated by removal with the cold wire snare, followed by cauterization with chloracetic acid.

So-called False Angioma of the Nose.—In the January number of *Pediatrics*, Dr. George H. Godson, of New York, reports a case of traumatic angioma of the nasal mucous membrane, the history of which reads like that of a semi-organized blood-clot which subsequently underwent absorption.

Microscopic examination of the growth, as in Dr. Glasgow's case, would have placed the diagnosis beyond doubt.

Trigeminal Neuralgia Relieved by Turbinectomy.—Mr. Walter G. Spencer showed to the Laryngological Society of London, January 12, 1898, a carpenter, forty-six years of age, who, a few days after an attack of influenza, had been suddenly seized with trigeminal neuralgia, which resisted treatment for some five months. On examination of the left nasal passage, although no definite disease was noticed, a severe paroxysm of the pain and itching occurred when the anterior part of the left middle turbinate was touched. These symptoms were not re-excited after the parts had been coated with a 20 per cent. solution of cocaine. There was no evidence of any other lesion.

The middle turbinate was excised, although it was not in contact with the septum. Nothing abnormal was found in the tissue removed, but from the

time of the removal the patient has had no pain, and has not required a narcotic, although at times there is slight itching at the terminal distributions of the fifth nerve of the face, the back of the eye and the nose.

Hypnotism in Nasal Obstruction.—Dr. Taptas, of Constantinople, reports (*Revue Hebdomadaire de Laryngologie, d'Otologie*, etc., January 29, 1898) a case of enlargement of the turbinates which was cured by hypnotic suggestion. The patient was a girl nineteen years of age, who had submitted to total hysterectomy two years previously. She was unable to breathe with her mouth closed for more than three respirations. The doctor advised treatment by cauterization, with subsequent removal of the posterior hypertrophies. Having, however, a few days previously relieved this patient, by hypnotic suggestion, of chronic constipation of several years' duration, he thought he would try what hypnotism would do for this lesion. So he commanded the patient to go to sleep, and to breathe through the nose freely. She obeyed, and continued to be able to breathe freely on coming to. The relief was permanent, notwithstanding she had been unable to breathe through the nose for a year.

[The record of this case should be taken to heart by those who mistake intumescence for hypertrophy and subject their patients to unnecessary mutilation. A purge or a series of purges, attention to the skin and kidneys, will often withdraw excess of blood from the distended turbinates, and save their subjects from unnecessary operation. Even a brisk run up-stairs will sometimes produce a temporary amelioration.]

Empyema of the Frontal Sinus.—Mr. Ernest Waggett exhibited to the Laryngological Society of London at its session, January 12, 1898, a patient upon whom he had performed Luc's operation five weeks previously for suppuration of the right frontal sinus of many years' standing. The incision through the skin followed the line of the eyebrow, and the trephining was practised immediately above the superciliary ridge. The sinus was completely cleared of all the mucous membrane which, throughout, was polypoid and bathed with pus. Attention was drawn to the advantages of carefully suturing the periosteum over the perforation made by the trephine, and of removal of the anterior end of the middle turbinate body. The cavity was irrigated by passing a fine flexible tube up through the drain-tube. The latter was removed on the thirteenth day. Pus had not been seen since the operation, and there were no subjective symptoms; depression of the bone could not be detected, and the scar of the skin was unnoticeable.

In the discussion Dr. Herbert Tilley considered the case as a good illustration of the value of incision through the line of the eyebrow, instead of the vertical incision in the middle line, and stated that the case was at least the second or third which had been before the Society in which the value of this incision was very evident.

Suppuration of the Maxillary Sinuses, with its Relation to Chronic Bronchitis.—Dr. Brindel, of Bordeaux, contributes a paper to the *Revue Hebdomadaire de Laryngologie, d'Otologie*, etc., February 5, 1898, illustrated by records of cases, and calling attention to the fact that chronic bronchitis

is often an extension from suppuration in the sinuses, and is permanently overcome by curing the original sinusitis.

Foreign Bodies in the Maxillary Sinus.—In a reprint from the *Monatschrift für Ohrenheilkunde* upon "Foreign Bodies in the Maxillary Sinus," DR. ZIEM, of Danzig, mentions two cases in which he removed canulas which were beyond the reach of forceps, by means of a steel probe, one end of which was slightly bent into a hook. This was placed through the canula, then drawn about 180 degrees around its axis and quickly withdrawn. In one case he succeeded with the first attempt, and in the other at the second attempt.

Dr. Ziem mentions the following additional methods of treatment: Introductions of electric magnet for steel canulæ; division of the alveolar process upon the foreign body with bent scissors; massive injections of fluid into the sinus to drive the foreign body through the nose, as has been successfully practised by Moure and others, and finally direct access by a large opening into the sinus from the canine fossa—a method which should not be practised but as a last resource.

Removal of a "Pressure Pouch" of the Œsophagus.—MR. HENRY T. BUTLIN, of St. Bartholomew's Hospital, reports (*British Medical Journal*, No. 1931) a second case of removal of a pressure pouch of the œsophagus, his first case having been reported with illustrated collateral material in the *Transactions* of the Royal Medical and Chirurgical Society in 1893.

The case in question was in a gentleman, aged seventy-three years, and supposed to be suffering from stricture of the œsophagus due to varicose veins in his throat, which veins had been burned and the uvula shortened by a throat specialist in large practice.

A demonstration of the pouch being perfect on examination under anæsthesia, the operation was performed at once, a long incision being made on the anterior border of the left sterno-mastoid, with its centre opposite the cricoid cartilage. The dissection was carried down between the trachea and the œsophagus on the inner side, and the great vessels on the outer side. The superior thyroid artery was tied, the omo-hyoid muscle divided, and the pouch was discovered lying behind the œsophagus, at its junction with the pharynx and projecting a little toward the left side. It was cut away from above downward, and the edges of the wound were brought together with fine silk sutures. No attempt was made to keep a tube in the œsophagus for feeding purposes, and a drainage-tube, instead of gauze, was inserted in the wound of the neck. In three weeks the patient was perfectly well, and the wound healed.

The case is illustrated by a drawing of the pouch, and is accompanied with a notice of six cases of the kind seen by Mr. Butlin, who calls attention to the lesion being frequently mistaken for stricture of the œsophagus, under which impression, as in two cases mentioned, useless gastrotomy has sometimes been performed, the real pathologic condition having been discovered only after the death of the subject.

The return of fragments of undigested food, hours after the meal, is one of the constant diagnostic symptoms.

[All of Mr. Butlin's patients have been men, in whom the symptoms were first noticed after forty years of age, but the present compiler remembers having seen at least one instance in a female subject, the wife of one of his medical friends.]

Pharyngeal Tuberculosis.—RETHI (*Annales des Maladies de l'Oreille, du Larynx*, etc., October, 1897) records a case in a young man, aged twenty-three years, who had been suddenly seized six weeks previously with violent dysphagia and pains in the left side of the neck, extending to the left ear. The disease had commenced in the left wall of the pharynx and presented an extensive ulceration reaching nearly to the orifice of the Eustachian tube, touching posteriorly the inferior portion of the tonsils and the posterior surface of the soft palate.

The case was not looked upon as promising much scope for treatment.

OTOLOGY.

UNDER THE CHARGE OF

CHARLES H. BURNETT, A.M., M.D.,
AURAL SURGEON, PRESBYTERIAN HOSPITAL, ETC., PHILADELPHIA.

Treatment of Attico-Mastoid Suppurations and Consecutive Intracranial Affections.—R. BOTEY (*Annales des Mal. de l'Oreille*, vol. xxiii., No. 11), after a careful discussion of the symptoms and indications in the above-named lesions of the ear and cranial cavity, formulates the following rules of action:

1. In acute antro-mastoiditis open the mastoid after the method of Schwartze. 2. In chronic affections expose the antrum and drum-cavity. 3. If the lesions are deep and the auditory canal wide, attico-antrectomy should be performed. 4. If the lesions are extensive and peripheral, antero-atticotomy (Stacke) should be performed. 5. Rapid intervention at the first symptoms of cerebral complication. 6. Never open the dura mater if it is not affected, but, after having carefully disinfected the meningeal surface, explore the nervous centres with a trocar and canula, 2 mm. in diameter, in order to prevent occlusion of the canula with a fragment of cerebral substance. 7. Make a perforation in the upper or the posterior wall of the antrum, after having opened the mastoid and the tympanum, in order to penetrate the cranial cavity (Wheeler and Broca), *but combine this with a perforation of the temporal bone at the lowest part of the squama, by making an opening a centimetre wide on the upper edge of the bony meatus, the former opening not being allowed to* extend more than a centimetre in width, for exploration of the middle cerebral fossa. 8. If the affection is very grave an exploratory puncture may be made through the lowest part of the squama. 9. No lavage; dressings every twenty-four or forty-eight hours, and filling the cavity with a bunch of very narrow rubber tubes.

Treatment of Otitic Complications.—HEIMAN (*Annales des Mal. de l'Oreille*, vol. xxiii., No. 2) has formed the following conclusions regarding the treatment of the serious complications of purulent otitis media: 1. The vital complications of otorrhœa still suffer from many wants in diagnosis and treatment. 2. In purulent otitis media, if there is no retention of pus, and yet general symptoms exist, like fever, debility, etc., or local lesions in the cerebrum, opening the cranial cavity is indicated in addition to the perforation of the mastoid. 3. Opening the cranial cavity is indicated even in the cases in which the surgeon does not find there the morbid products he expects to discover; furthermore, this operation diminishes the intracranial pressure as well as the conditions favoring the absorption of infectious elements. 4. When one suspects the existence of a thrombus in the lateral sinus, it becomes necessary to puncture and aspirate the contents of the sinus, after opening the skull cavity. This latter part of the operation is of very great importance in diagnosis, and decides in most cases the necessity of continuing the operation on the sinus. 5. Exploratory puncture and even incision of the sinus will not entail general infection if the operation is surrounded with aseptic precautions, and it is not, furthermore, injurious when infectious conditions already exist. 6. Clinically it is necessary to make a distinction between two forms of pyohemia, viz., that with thrombus and that without thrombosis of the sinus. For operative procedure, pyohemia with thrombus of the lateral sinus offers the most important consideration. 7. Thrombotic pyohemia arises usually from pyohemia without thrombosis. The two forms occur if the infectious sources in the organ of hearing, and especially those in the cranial cavity, are not removed promptly, and the absorption of these matters suppressed. The development of the two forms of pyohemia often dates from the moment when the absorption of the infectious masses begins. 8. Pyohemia without thrombosis generally terminates favorably under the application of a rational therapeusis, and sometimes even without treatment. 9. Pyohemia with thrombus generally ends in death. Prompt and efficient operative interference, however, saves a certain number of cases. 10. The selection of the time of operating in vital complications of purulent otitis, especially when symptoms of general infection arise, is often difficult. When the surgeon dare wait without disadvantage to the patient, it is better not to operate until he is assured in his diagnosis.

[Most of the latest authorities agree in their opinion that it is better to explore in order to confirm the diagnosis of the presence of a suspected intracranial lesion than to wait for symptoms to confirm the diagnosis, when it will be generally too late for any operation to do any good.]

Eucaine as a Local Anæsthetic in the Ear.—HORNE and YEARSLEY have formulated their experiences with eucaine hydrochloride in relation to the surgery of the throat, nose, and ear (*British Medical Journal*, No. 1926). The strengths of the solutions used were 2, 5, and 8 per cent., applied by instillation in the ear, swabbing in the throat, and on pledgets of cotton-wool in the nose. The anæsthesia was found as efficient as that of cocaine. In three cases out of thirty-two unpleasant after-effects were noticed; but "these could be fully explained by other factors present." The effects upon the pulse appeared to be *nil* or very slight. These observers found that instead of

hyperæmia of the turbinates, this drug produced ischæmia of these parts, but not ischæmia comparable to that of cocaine. In the ear it seems to have acted as well as cocaine, but no better.

Goutiness in its Relations to Ear Diseases.—A. H. BUCK starts out with the "premise that a patch of eczema, of spontaneous development, signifies the existence, in the individual possessing it, of a gouty diathesis." . . . . "As it happens, the floor of the external auditory canal is apt to be the very first spot on the surface of the body where an eczematous inflammation develops." This condition the author claims "constitutes a valuable guide-post, pointing as it does, at a very early stage, to the existence of that disturbed state of the metabolic processes to which the term goutiness, or a gouty diathesis, is applied." In the cases related it is shown "how different localities or different tissues are involved at different times." At one time "it is the skin of the external auditory canal and auricle; at another time, the dermoid surface of the tympanic membrane is involved, as well as the walls of the canal; at still another, the adjacent bone tissue is affected; and, finally, at a fourth, the disease locates itself in the mucous membrane of the tympanic cavity, and particularly in that part of it which borders upon the two fenestræ." The treatment applied to these lesions, as set forth minutely in the notes of the cases appended to the paper, consisted largely in local applications to the diseased territories in the ears, with combination in some cases of Carlsbad salts. More or less relief was given in this way; but if patients are of irregular habits of living, which they will not give up, relapses in ear-lesions must occur.—*Transactions of the American Otological Society*, vol. vi., Part IV.

Ear-complications in Influenza.—EAGLETON claims that cases of catarrhal otitis complicating influenza, and going on finally to suppuration, may present one of three conditions, that "are probably due to the influence of the presence of Pfeiffer's bacillus:" 1. "Distinctive types of hemorrhagic otitis." 2. "Primary mastoiditis or periostitis before involvement of the middle ear, due apparently to direct infection by the bacillus, and not to extension from the nasopharynx." 3. "Rapid caries and necrosis of ossicles or mastoid, this last being of very frequent occurrence." He also thinks that "the presence of the influenza bacillus exerts a very unfavorable influence on the bony structures of the ear, often converting apparently simple cases of acute suppurative otitis into very malignant ones, with rapid destruction of bone," not only in the drum-cavity, but in the mastoid.—*Transactions of the American Otological Society*, vol. vi., Part IV.

[We have never observed any specific tendency toward virulency nor malignancy in otitis media occurring in epidemic influenza. The course of otitis media in this disease, as in all others, depends upon the judicious, non-irritative treatment of the primary inflammation in the nose and ear.]

Phosphorus Necrosis of the Temporal Bone.—This rare disease has been observed in one instance and reported by H. V. WÜRDEMANN. The patient was a man, aged fifty-eight years, employed in a match factory. Two years after the development of phosphorus necrosis of the upper jaw on the left side the left mastoid began to be painful, and, finally, about a year later,

pain in the ear was felt in addition to the mastoid pain, and finally the ear discharged some foul muco-pus. The membrana flaccida was found perforated. For the necrosis about the mastoid and the chronic purulency in the attic region, antrectomy was performed by Würdemann, and relief followed for five months. Then another operation on the mastoid and antrum was performed, and relief for nearly a year ensued, when again the ear demanded an operation on the mastoid. "The mastoid was chiselled and drilled open, the external layer being found hard and cancellous, under which there was found a pasty mass of necrosed bone from which exuded greenish, fetid pus. The posterior wall of the canal was soft and friable and readily removed by the spoon. It was found that the superior wall had been largely reproduced, and thereupon the triangular piece of bone between the attic and the tympanum was removed and granulations taken away from the tympanic attic." Immediately after this last operation the wound, after the second dressing, was free from pus.—*British Medical Journal*, No. 1926.

Symptoms Demanding Operation on the Mastoid.—BACON looks upon elevation of temperature, though slight, combined with tenderness on pressure over the mastoid process, in a case of acute otitis media of ten days' standing, as characteristic of mastoid disease. "Pressure should always be made on both mastoids, however, as occasionally such pressure causes pain in a healthy mastoid." . . . "Bulging of Shrapnell's membrane, with drooping of the posterior and upper cutaneous lining of the external meatus, are to my mind absolute symptoms of mastoid involvement, and I believe that in such it is always necessary to perforate the mastoid cells." If mastoid symptoms, pain, etc., in connection with chronic purulent otitis media, do not yield to local and general antiphlogistic remedies, there should be no hesitation in making an exploratory opening in the mastoid, when we shall generally find quite as much if not more disease than we had expected.—*Transactions of the American Otological Society*, vol. vi., Part IV.

Anatomical Points Bearing on Operations on the Temporal Bone.—H. L. MORSE has shown that in Schwartze's operation on the mastoid, antrum, and middle ear there is less likelihood of wounding the lateral sinus, the facial nerve, the tendon of the stapedius muscles, the stapes, and the horizontal semicircular canal than there is in Stacke's operation in the same region, because in the latter procedure the field of operation, especially at the beginning, lies nearer these delicate structures than it does in the Schwartze operation. The latter is therefore preferable whenever it can be employed. However, in cases of sclerosis of the mastoid, or where the lateral sinus projects so far forward as to lie just beneath the mastoid cortex, the Stacke operation or a similar one, such as Hartmann's, Küster's, or von Bergmann's, is preferable, because they proceed at once further forward and nearer the seat of disease, even if they possess the disadvantage of a limited field of operation among delicate tissues.—*British Medical Journal*, No. 1926.

Perisinal Abscess with Thrombosis of the Lateral Sinus.—ALDERTON reports a case of perisinal abscess with thrombosis of the lateral sinus

After opening the mastoid cavity and attempting to reach the antrum he came immediately upon the sigmoid groove for the lateral sinus. Pus welled up through the opening thus made, the pus having dissected away the sinus from the bony wall. "As the sinus lay in the tract usually followed to reach the antrum, as the sinus pulsated, and as it was thought that perhaps outlet given to the pus might be all that was needed, it was decided to wait twenty-four hours and watch the progress of the case before proceeding further."

The next day it was decided to open the sinus. This was found to contain "puriform fluid composed of broken-down clot. The sinus wall was then cleansed of infectious material, and with the probe and curette the clot was dislodged, both above and below, until free hemorrhage was induced." The antrum was not entered at this time. The patient developed metastatic pneumonia, and died six days later. In reviewing the case, Dr. Alderton says: "This case emphasizes the necessity of gaining access, however difficult, to the antrum in all cases of mastoid, sinus, or brain involvement. It does not do to trust to nature after having given exit to the pus. If the antrum had been opened by chiselling away the posterior-superior wall of the external canal and the adjacent bone, this patient would undoubtedly have been in a better position for recovery. To the writer it also seems to emphasize the desirability of ligating the internal jugular in the neck in all cases of septic thrombosis of the lateral sinus, to prevent the extension of sepsis."—*Transactions of the American Otological Society*, vol. vi., Part IV.

OBSTETRICS.

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.,

PROFESSOR OF OBSTETRICS AND DISEASES OF INFANCY IN THE PHILADELPHIA POLYCLINIC; CLINICAL PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE; CLINICAL PROFESSOR OF DISEASES OF CHILDREN IN THE WOMAN'S MEDICAL COLLEGE; VISITING OBSTETRICIAN TO THE PHILADELPHIA HOSPITAL, ETC.

Posterior Rotation of the Occiput.—MOTTA (*Archiv für Gynäkologie*, 1879, Band liv., Heft 3) reports eighty-three cases from the Dresden clinic, and draws conclusions from them.

As regards the cause of this abnormality, he is inclined to believe that the form of the uterus, the direction of its axis, and the direction in which uterine contractions act exert the greatest influence. Of his eighty-three cases thirty-nine were primiparæ and forty-four multiparæ. In eighty-one of these cases some abnormality in the shape of the pelvis existed, the pelves being contracted. Early rupture of the membranes favored this abnormal rotation, as 48 per cent. of his cases had the waters break prematurely. This fact and the abnormal size of the pelves lead to the conclusion that a disproportion between the fœtal head and the pelvis is the primary cause of the abnormal rotation.

As regards treatment, attention is drawn to the difficulty often experienced in the early application of forceps. On the other hand, expectant treatment is attended with considerable risk to mother and child. Motta's cases were treated by version in thirty-three; craniotomy in twenty-eight; the forceps in five; Cæsarean section in four; and symphyseotomy in one; while twelve terminated spontaneously. Of the children, thirty-nine were born living. All the mothers recovered. Motta advises the careful preservation of the bag of waters, and the use of elastic dilators if necessary. When delay cannot be practised, version should be performed if the head has not engaged. When dilatation is not complete and the membranes have ruptured early, the safest method of delivery in many cases will be found in Cæsarean section. The use of forceps will occasionally succeed if combined with Walcher's position.

[In the paper just cited the author has overlooked the considerable proportion of cases in which the occiput rotates in front without interference. Two groups of cases, aggregating over six hundred, may be found in the literature of the subject, in which 98 per cent. resulted in spontaneous anterior rotation of the occiput.]

Syncytioma Malignum.—GEBHARD reports, in the *Zeitschrift für Geburtshülfe und Gynäkologie*, 1897, Band xxxvii., Heft 3, two cases of this form of tumor, and describes the growth in detail. His first patient was a multipara who was seized with bleeding seven weeks after her last confinement. The uterus was as large as a fœtal head, and its interior contained a soft, irregular growth. The uterus was removed through the vagina, and examination of the tumor showed it to be derived from the decidua, and malignant in its tendency.

The second case was also a multipara who had suffered from pain in the back and abdomen, with irregular bleeding, for two weeks. On examination a mole was found and removed, the patient making a good recovery. As hemorrhage recurred, a curetting was done for diagnosis, and a malignant syncytial tumor was found to be present. The uterus was removed through the vagina. Both of these patients made good recoveries.

Microscopic examination of each case gave the characteristic appearance. He also describes a third case in which a tumor of the vaginal-wall was present, which was removed, the patient subsequently perishing. Post-mortem examination revealed multiple metastases, the original growth having been malignant.

A careful study of the third case leads the writer to class it as a syncytioma.

He also describes a carcinoma whose cells took on the same peculiar and irregular wandering growth which is peculiar to syncytioma. It is evident that a most careful examination is required to make a positive diagnosis between the two.

Incision of the Fundus in Cæsarean Section.—In the *Centralblatt für Gynäkologie*, 1898, No. 9, MÜLLER states that he has, in a number of cases, incised the uterus, not across its fundus, but at the fundus in its long axis. He has met with little hemorrhage. The child could be rapidly extracted, and the uterus is easily closed by various layers of continuous catgut sutures.

He leaves his stitches within the uterus, having no hesitation in sewing through the uterine decidua. He thinks that hemorrhage is much less likely after this proceeding. He has employed this method of operation for six years, with excellent results.

Fifty-one Cases of Placenta Prævia.—JARDINE (*Glasgow Medical Journal*, 1898, No. 1) describes in detail a series of cases of hemorrhage from placenta prævia. Among others he cites an interesting case in which De Ribes' bag burst while in use. The fluid separated the placenta and brought about a fatal hemorrhage. The most successful treatment in these cases is delivery performed as promptly as possible.

Extra-uterine Gestation.—In the *British Medical Journal*, 1898, No. 1935, MAYO ROBSON reports seventeen cases of extra-uterine gestation, and draws some conclusions from them.

As regards diagnosis, there was no difficulty in the acute cases. Special symptoms of value were a superficial dulness on percussion over the pubes and in either flank, with resonance on deeper percussion. Also a thrill in the same region, although fluctuation could not be felt. On turning the patient over, dulness in the upper flank persisted for some time, but gradually disappeared, a sign only present when blood is in the peritoneal cavity. Dysmenorrhœa and pain are often present. In old cases a painful tumor near the uterus, metrorrhagia, and the passage of membrane are important symptoms.

As regards treatment, there is no safety except in operation.

Malignant Disease in Pregnancy.—In the *Scottish Medical and Surgical Journal*, 1898, No. 2, FOTHERGILL reports the case of a woman, aged thirty years, who died of a malignant pelvic growth. There were no metastases. The growth was composed of alveoli separated by bands of fibrous tissue. It was a malignant adenoma arising from the glands of the cervix during pregnancy. He describes also a case in which a foreign growth involved the labia and also the pelvic organs. This was found to be a round-celled sarcoma. The history given by the patient was that she was well until her last confinement, when she first noticed an offensive discharge. On further investigation, the physician who delivered her stated that at labor malignant disease was so far advanced that it was necessary to deliver by craniotomy and afterward sew up the cervix.

A third case is described in which tissue removed from the uterus proved to be round-celled sarcoma resembling very closely a syncytioma, occurring in a uterus which was fibroid.

In each of these cases it would have been easy to arrive at the conclusion that these tumors were syncytial and directly derived from the uterine decidua. Fothergill urges that the greatest care be taken in diagnosis, that errors may be avoided.

Pulmonary Congestion and Œdema During Pregnancy and Labor.—DUPLANT (*Gazette Hebdomadaire*, 1898, No. 9) reports three cases of cardiac disease in pregnancy and labor, accompanied by acute engorgement and œdema

of the lungs. He concludes, from a study of these cases and a review of the literature, that acute congestion may complicate pregnancy and labor independently or in connection with cardiac disorders. When acute pulmonary œdema occurs alone, labor does not aggravate it, and increases the work of the heart but very little. Both heart and lung failure, however, may develop rapidly after the labor. Rupture of the utero-placental circulation aggravates the œdema, as does the evacuation of the uterus, the hemorrhage of labor, and the suppression of the utero-placental circulation which throws the heart into a condition of asytole. The acute congestion occurring during labor seems due to a reflex of uterine origin and to a modification of the blood during pregnancy. To avoid such complications, when the heart is threatened with asystolic failure, or when acute œdema suddenly develops, labor should be immediately brought on. The bleeding which often occurs in labor is an advantage in these cases.

Antitoxin in Puerperal Sepsis.—CLARK (*Boston Medical and Surgical Journal*, 1898, No. 2) reports two cases of puerperal sepsis treated by antitoxin.

The first was a multipara who had pelvic trouble after a former labor. Her labor was normal. She developed high fever, with soft and flabby uterus. The usual treatment of septic infection proving ineffectual, and septic absorption proceeding, 20 c.c. of antistreptococcus serum were injected. Immediate improvement followed. The dose was repeated, and convalescence ensued.

The second case was a multipara who had an easy labor. She developed diphtheritic infection of the genital tract, cultures from the vagina showing Klebs-Löffler bacilli and staphylococci. Double antidiphtheritic and antistreptococcic serum was given in dose of 40 c.c., which was repeated on the following day. Local disinfection of the uterus was continued. The patient made a tedious recovery.

In both these cases local disinfection, free purgation, and stimulation were ineffectual, and the use of serum was followed by prompt improvement.

Heart Disease and Pregnancy.—PINARD, in a clinical lecture (*Medical Press*, 1898, No. 3065), described two cases of valvular heart disease in pregnant women, and drew the following practical conclusions: Pregnancy of itself does not provoke heart lesions. In women who have cardiac disease and become pregnant, if the kidneys are sound, compensation is established.

The treatment of these cases is exceedingly simple—absolute rest, milk-diet after the fourth month, and digitalis in infusion at intervals. Should syncope and complications arise, empty the uterus promptly and bleed.

GYNECOLOGY.

UNDER THE CHARGE OF
HENRY C. COE, M.D., M.R.C.S.,
OF NEW YORK.

Operations for Prolapsus Uteri.—SANGER (*Centralblatt für Gynäkologie*, 1898, No. 2) in an elaborate paper on this subject describes an operation which he has practised with success. A suture is introduced just behind the portio in the median line of the posterior vaginal wall. After thorough dilatation of the sphincter ani, the left forefinger (protected with a rubber cot) is introduced into the anus and inverts the recto-vaginal septum as much as possible. A vertical incision is made in the posterior vaginal wall with a scalpel, and crescentic, or V-shaped, flaps are dissected off from its lower end with scissors, their size varying with the amount of redundant tissue. While these flaps are held apart with forceps, the upper edges of the wound are detached in the same manner. When the peritoneum is reached, it is carefully pushed upward with the finger or a gauze-pad; the rectum is separated with the same care. The edges of the levator ani are also detached as low down as the vulva.

The flaps are now cut away, bleeding vessels being caught with forceps. The denuded surface resembles that made in Hegar's operation, except that it extends higher and also approaches nearer to the anus, and extends laterally into the para-vaginal connective tissue. The upper sutures (silk) are passed in the usual manner, buried catgut being used if there is an extensive rectocele. Toward the lower end of the wound the vaginal edges alone are included in the sutures. The results of this operation, when properly performed, have been entirely satisfactory.

Does Cauterization Prevent Infection?—BRINK (*Ibid.*) endeavors to answer this question by experiments on animals. Guinea-pigs were etherized, the abdomen opened, and an eschar made on the peritoneum with a glowing platinum wire. Without disturbing the wound, it was touched with another wire dipped in a staphylococcus culture. The wound was then closed and sealed with iodoform-collodion. Two days later the animals were killed, the abdomen excised with a cautery-knife, and the eschar removed with aseptic precautions. In all the cultures made from the tissue immediately beneath the lesion colonies of staphylococcus aureus were obtained, which were inoculated with the usual results. In animals killed from six to ten days after the primary inoculation the micro-organisms were found in the deeper tissues.

The writer infers not only that an eschar does not prevent the penetration of pathogenic organisms into the subjacent tissue, but that it probably fails to prevent re-infection of raw surfaces during operations for cancer. Hence the results claimed for igni-extirpation of the cancerous uterus are open to considerable doubt.

Cystitis of Infectious Origin.—KARGER (abstract of monograph in *Centralblatt für Gynäkologie*, 1898, No. 2), from a careful analysis of forty-six cases, arrives at the conclusion that cystitis (with certain rare exceptions of chemical or toxic origin) is always due to micro-organisms, the bacterium coli commune being the most common. The mucosa of the bladder, however, must previously be in a condition favorable to infection. The existence of so-called "catarrhal cystitis" is doubtful. Ammonuria is of secondary importance; in the majority of cases the reaction is acid. True gonorrhœal inflammation of the bladder is always caused by Neisser's coccus.

The prevention of cystitis lies in the maintenance of absolute asepsis of the meatus urinarius, as well as of the urethra. A four per cent. solution of nitrate of silver is the best remedy in inflammation of the bladder.

Prolapse of the Bladder in the Female.—KLEINWÄCHTER (*Zeitschrift für Geb. u. Gynäkologie*, Band xxxiv., Heft 2) has collected only eighteen cases from the literature, to which he adds another. Nine occurred in children, eight cases being those of complete prolapse through the urethra. The accident occurred suddenly, in consequence of violent abdominal pressure, or gradually. The diagnosis is not usually difficult.

The treatment consists in replacing the inverted organ under anæsthesia, after which pressure is maintained against the neck of the bladder by means of tampons, the patient being kept perfectly quiet for several days. In adults the catheter should be used regularly. In the latter a pessary may later be substituted for the tampon, or, if this fails to prevent a recurrence of the prolapse, ventro-fixation of the bladder may be practised. Of the cases reported, eleven patients recovered without an operation; four of the others died, two of uræmia.

Therapeutic Effect of Mechanical Irritation of the Uterus.—REINECKE (*Münchener med. Wochenschrift*, 1892, No. 52) believes that one of the most valuable results of massage of the uterus is the causing of contractions, with resulting changes in its shape and size. As Arendt has shown, in many cases of retroflexion without fixation, reposition is rendered difficult purely on account of the size of the uterus. Massage at the point of flexion, with one finger in the rectum and the other in the vagina, produces contractions, in consequence of which the isthmus hardens and the corpus uteri is felt to rise perceptibly. If this treatment is continued, reposition can easily be effected. Subinvolution and venous congestion are also greatly relieved by this method of massage.

Atrophy of the Uterus Following the Introduction of Steam.—BARUCH (*Centralblatt für Gynäkologie*, 1898, No. 5) reports a case which shows that the intra-uterine use of steam in cases of hemorrhage is not free from evil results. The patient, aged twenty-seven years, had a return of the menses four months after delivery, the flow continuing for eight weeks. This was at once checked by steam, and did not return, the patient suffering from the usual symptoms of the climacteric. Tablets of ovarian extract were given for some time, which caused severe abdominal pains, but no reappearance of the menses.

On examination two years after the labor the uterus was found to be small and hard, and the externum obliterated by cicatricial tissue, so that a probe could not be passed. The ovaries could be felt indistinctly.

The Abdominal Reflex in Women.—BODON (*Ibid.*) believes that the abdominal reflex possesses a certain diagnostic value in doubtful cases. In pelvic cellulitis the reflex is not affected, while in pelvic peritonitis it is either diminished or weakened upon the affected side. Hence when a doubtful tumor is discovered on one side of the pelvis, and no difference in the belly reflex is found on the affected side, it may be inferred that the mass is situated in the pelvic connective tissue. If, on the contrary, the reflex on the affected side is weakened or absent, it is probable that the peritoneum is involved—*i. e.*, there is an exudate around the tube and ovary, or a pelveo-peritonitis. In acute cases the writer often observed that when the abdomen was lightly stroked over the affected side, the patient was suddenly seized with a violent pain, which was entirely out of proportion to the mechanical irritation. This is probably explained by the movements of the inflamed peritoneum caused by slight, invisible contractions of the abdominal muscles. In one case the pain was felt on the healthy side.

From further experiments upon the round ligaments the writer concludes that these are the analogues of the cremasters in the male.

Pregnancy and Parturition Following Amputation of the Cervix Uteri.—AUDEBERT (*Annales de Gynécology et d'Obstétrique*, January, 1898) concludes an interesting article on this subject as follows: Amputation of the cervix exercises an important influence upon subsequent pregnancy, frequently leading to abortion or premature delivery. During labor the membranes are prone to rupture prematurely, while in some cases the cicatricial tissue opposes an absolute impediment to dilatation, requiring multiple incisions to overcome it.

Intra-vaginal Pressure as an Aid in Abdominal Operations.—NEUGEBAUER (*Centralblatt für Gynäkologie*, 1898, No. 5) conceived the idea of assisting the removal of intra-pelvic neoplasms by elevating the pelvic contents by pressure exerted through the vagina by means of a colpeurynter. Before operating upon a case of extra-uterine pregnancy he introduced the rubber bag and distended it with water to its utmost limit. On opening the abdomen he was surprised to find that the tumor, which was previously situated deeply within the pelvis, now appeared immediately beneath the wound and was easily removed. As the colpeurynter was emptied, the pelvic organs sank downward to their normal position.

Appendicitis in its Relations to the Pelvic Organs.—KRÜGER (*Deutsche Zeitschrift für Chirurgie*, Band xlv., Heft. 3 u. 4) emphasizes the importance of the differential diagnosis between appendicitis and disease of the adnexa. Acute puerperal parametritis may begin in the same manner as perforation of the appendix, but the symptoms are less severe, those of diffuse peritonitis being absent.

It is more difficult to distinguish between perforation of the appendix and

the rupture of a pus-tube or ovary. If recovery takes place, the parametritis and para-typhlitic exudates can usually be diagnosticated by their characteristic shape and position.

Diseased adnexa are generally recognized from their close relation to the uterus. When these are associated with appendicitis the diagnosis may be exceedingly difficult.

Alexander's Operation.—Simões (*La Gynécologie*, 1897, Nos. 3 and 4) concludes an elaborate paper on this subject with the following summary: The operation should only be performed in cases of movable uterus. Since metritis and prolapsus usually accompany the retro-deviation, curettement, amputation of the cervix, and colpo-perineorrhaphy should supplement Alexander's operation in cases of chronic retroflexion. Extra-peritoneal shortening of the round ligaments is preferable to all the other methods of supporting the uterus in its normal position, as it is the most simple and rational. Finally, statistics have shown that pregnancy is least likely to be interfered with after this operation.

PÆDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D.,

OF PHILADELPHIA.

ASSISTED BY

THOMPSON S. WESTCOTT, M.D.,

OF PHILADELPHIA.

The Clinical Variability of Rubella.—Péron (*Revue mensuelle des Maladies de l'Enfance*, October, 1897) records three cases of rubella in a family, one of which, in the person of the father, aged forty-five years, presented almost a typical picture of severe measles.

The first patient affected was a son, aged eleven years, who, after three days of mild prodromes, presented a rapidly spreading typical rubellous eruption, with enlargement of the post-cervical lymphatic chains, slight fever, secondary sore-throat, slight hoarseness, coryza, and infrequent cough. The attack terminated on the third day.

Fourteen days after the appearance of the son's eruption the father was taken ill, with violent headache, lassitude, and chilly sensations. Then followed violent attacks of sneezing, hoarseness, and cough. The latter became very frequent and distressing, and disturbed sleep as markedly as in the prodromal stage of measles. Toward evening of the fourth day the symptoms increased, the patient took to bed, passed a bad night, and awoke with a confluent and generalized eruption. There was slight cervical adenopathy. With the appearance of the eruption there were abundant coryza, lachrymation, and conjunctival injection. Upon the uvula was seen a typical rubellous mottling.

Temperature 103° F. The eruption lasted four days, and on the second day the morning temperature was 101.4° F. The eruption disappeared progressively; but for fifteen days, at least, the voice remained hoarse and the cough frequent and distressing. Convalescence was slow. The tongue was thick and coated for several days. Cerebral and muscular activity was not regained for several weeks.

On the eighteenth day after the son's eruption appeared, the youngest child, aged four years, was seized with slight illness, and in a few hours showed a slight rosy blush of the face. Upon the body was noted only a slight rose coloration of the buttocks and loins. The ganglia of the neck were slightly tender on both sides. There was no buccal eruption, and temperature was 99.5° F. The whole condition had passed away by evening of the same day.

It is further stated that measles had not occurred in this family at the time of the illness above described; but the father had passed through an attack in his childhood, and, moreover, typical measles was subsequently contracted by the two boys and another brother during the same year.

In support of the rubellous nature of the father's attack the author calls attention to the period of incubation of seventeen days in his case, and of eighteen days in the younger boy's, and to the brusque appearance and rapid generalization of eruption in both these patients.

The Therapeutic Value of Laparotomy in Tuberculous Peritonitis.—MONTI (*Wiener klinische Wochenschrift*, 1897, No. 42, S. 935) concludes a paper on this subject before the Twelfth International Medical Congress with the summary:

1. Tuberculous peritonitis, with serous exudate, can be cured by internal treatment, though much less frequently and always more slowly than by laparotomy.

2. In such cases operation gives very good results, but relapses and subsequent outbreaks of tuberculous processes in other parts of the body are, nevertheless, not necessarily prevented.

3. In tuberculous peritonitis, with extensive adhesion of intestines or coincident swelling of the mesenteric glands, with which there is little fluid exudate, the results of operation are not satisfactory, and may lead to an unfortunate termination.

4. Experience up to the present is too slight to determine with certainty the value of laparotomy in the treatment of tuberculous peritonitis, except in cases with serous effusion and without extensive adhesions of the bowels or swelling of the mesenteric glands, in which good results may be expected.

Unilateral Secretion of Tears in Facial Paralysis.—EMBDEN (*Société de Médecine de Hambourg*, October 29, 1897) reported the observation of a girl of four years, received into the hospital for a fracture of the skull, whose recovery was followed by a right facial palsy, with coincident paralysis of the uvula. Final examination at time of discharge showed the following condition:

There was palsy of the three branches of the right facial with the signs of degeneration. Crying was attended by secretion of tears from the left eye only, while the right eye remained dry. Redness of the face and increased

nasal secretion attending the act of crying remained localized to the healthy side. The functions of all the other cranial nerves remained normal. This observation, the author thought, seemed to support the view of Goldzieher, who considered the facial nerve to possess a secretory function.

New Researches upon Arrest of Growth and Infantilism.—Van den Corput (*Académie de Médecine de Belgique*, October 30, 1897) made a report upon the memoir of Hertoghe, of Anvers, in which that writer contends that arrests of growth, even when not myxœdematous in origin, such as are due to rhachitism, hyperazoturia, congenital syphilis, onanism, and other conditions, are amenable to thyroid treatment. He goes on to show that all arrested growth is dependent primarily upon an alteration of the thyroid gland. He distinctly asserts that the deleterious influences which retard growth exert their earliest effect upon the thyroid gland, which, as variously affected in its functions, produces, according to the degree of the lesion, obesity, rhachitism, chondro-drystrophy, or, finally, myxœdema more or less complete.

Arrest of growth by toxic substances, like tobacco and alcohol, he believes, is brought about in the same way through the functions of the thyroid gland. The nature of infantilism is thus considerably simplified.

Hertoghe has also studied the influence of the thyroid upon the development of the reproductive organs, and concluded that hypertrophy of the thyroid is the first sign of beginning puberty, and is indispensable to the adequate development of the sexual organs at puberty. According to this view, thyroid feeding finds a distinct indication in retarded puberty. [This paper, of which so far only a brief abstract has reached us, seems to open up a wide and fertile field for investigation.]

Transmission of the Power of Typhoid Agglutinative Action through the Milk.—Landouzy and Griffon (*Société de Biologie*, November 6, 1897) reported the observation of a woman of nineteen years, ill of typhoid fever three months after her confinement. The disease was of moderate severity and the serum reaction was positive. The child, which she had been nursing, appeared to be in perfect health. Examination of its blood showed that the agglutinative power was quite distinctly marked. This transmission of the agglutinative action of the blood-serum from mother to offspring has already been observed in mice by Widal and Sicard. The case recorded, therefore, proves that the same fact may be predicated of the human animal.

The Broncho-pulmonary Complications of Measles.—Hutinel (*Presse Médicale*, 1897, No. 38, p. 205) presents an interesting study of this subject, which may be epitomized as follows:

A simple infection of the bronchial mucous membrane, due to the usual unhygienic causes in association with the ordinary pyogenic bacteria, may, under certain conditions, become contagious when the virulence of these germs has been greatly increased. For example, if a child already suffering from streptococcic broncho-pneumonia is admitted to a hospital ward containing children with simple measles, the latter cases quickly become complicated, more or less seriously, with the same pulmonary lesion.

A child who previously to his measles has had a broncho-pulmonary infection may have a reappearance of this infection with the outbreak of measles, even though he had been apparently well of the precedent trouble for some little time. The previous existence of actual broncho-pneumonic lesion is not necessary, provided the infection itself has existed. Taken in its widest sense such pre-existent infection embraces the subjects of former tuberculosis, those that have had simple or specific catarrhs or localized pharyngeal infections, and finally those old hospital sojourners whose mouths and nasopharynges have had a chance from their surroundings to become infected with virulent microbes.

The crowding together of children increases the virulence of germs and the number and gravity of the infections. Frequently one observes the progressive aggravation of measles in isolating wards which cannot be frequently disinfected. Cases usually enter these pavilions in successive series. The first are usually benign cases; in the second series the fever falls less promptly, and more or less serious complications are noted; with the third series broncho-pneumonias often appear. These at first do not seem severe, but soon the severity of the complication increases, and fatal cases or subacute broncho-pulmonary infections become common.

It is to be observed that not only pre-existent infections of the respiratory passages may cause broncho-pneumonia in rubeolous patients, but the infection may be heteromorphous. A child may present a more or less grave cutaneous infection; following a measles he has a chance of infecting the lung. An intestinal or buccal infection may act in the same way.

Clinically the broncho-pulmonary complications of measles present three principal types: 1. A very acute form developing as a suffocative catarrh; 2. An acute form having all the characters of broncho-pneumonia; 3. A subacute or delayed form, suggesting tuberculosis, which may be called a pseudo-tuberculous broncho-pneumonia.

In order to avoid the broncho-pulmonary complications of measles it is therefore necessary to realize as much as possible the asepsis of rubeolous patients and to disinfect the quarters in which such patients are cared for.

Gangrene of the Soft Parts of the Thorax Following Measles.—WUNDER (*Münchener medicinische Wochenschrift*, 1897, No. 20, S. 536) reports the case of a girl, aged two years, who, on the tenth day of an attack of measles, presented a plaque of gangrene the size of the palm of the hand upon the right lateral aspect of the thorax, below the axilla. The periosteum of the subjacent ribs was laid bare, with a part of the great pectoral muscle. Cure resulted without functional impairment after a large abscess had developed in the internal aspect of the arm facing the gangrenous area.

The author attributes the localization of gangrene to the habit which the child had of lying on its right side. The compression had produced thrombosis of the long thoracic artery, with consequent gangrene of the tissues, whose power of resistance had been weakened by the disease.

Contribution to the Pathology of Pertussis.—Reasoning from the analogy of many other infectious diseases, FRÖHLICH, of Breslau (*Jahrbuch f. Kinderheilkunde*, 1897, Bd. xliv.), has succeeded in discovering that the blood

of children suffering from whooping-cough shows an enormous increase in the leucocytes, which is especially marked in the third or fourth week—that is, at the maximum of intensity of the disease. This leucocytosis is principally due to the lymphocytes, which are especially increased as compared with the polynuclear cells and the eosinophiles. In some of the cases this lymphocytosis reached 50 or 60 per cent., an unusually high degree as compared with other forms of leucocytosis, in which ordinarily a proportion of only 15 to 20 per cent. is reached. In these cases, however, the age of the patients must be taken into account, since leucocytosis with predominance of lymphocytes is, according to Weiss, very common in childhood.

The cause of leucocytosis in pertussis, Fröhlich thinks, can be explained by the fact demonstrated by Blumenthal, that there is a constant increase in the amount of uric acid excreted in the urine of these patients. Moreover, Horbaczewski has shown that in infectious diseases there is a constant relation between the exaggerated elimination of uric acid and the increase in the white corpuscles. The author suggests that the knowledge of the existence of leucocytosis in pertussis can be of practical value in the diagnosis of doubtful cases, especially where the clinical picture is incomplete, and in preventing children with a spasmodic cough, which is not really pertussis, from being exposed to the disease among other children with well-marked and undoubted clinical symptoms.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE DIRECTION OF

W. T. COUNCILMAN, M.D.,

SHATTUCK PROFESSOR OF PATHOLOGICAL ANATOMY, HARVARD UNIVERSITY.

AND

F. B. MALLORY, M.D.,

ASSISTANT PROFESSOR OF PATHOLOGICAL ANATOMY, HARVARD UNIVERSITY.

The Bacteriology of Whooping-cough.—The results of investigations on this subject have been quite contradictory until within the last few months, during which period three investigators have published results which practically agree. DEICHLER (*Zeitsch. f. Wissensch. Zool.*, 1887, Bd. xi.) described a protozoan which he found constantly in the sputum of whooping-cough. Later KURLOFF (*Centralbl. Bakt. u. Parasitk.*, 1896, Nos. 14 and 15, Bd. xix.) described flagellate bodies. AFANASSIEFF (*St. Petersburger med. Woch.*, 1887) and SENTSCHENKO (*Wratsch*, 1887) described a bacillus which they found constantly in the sputum. M. COHN and H. NEUMANN (*Archiv f. Kinderheilk.*, 1893, Bd. xvii.), after a prolonged study, came to a negative conclusion in regard to the bacillus, considering it as a saprophyte normally present in sputum, and described a diplococcus which they had met with constantly. About the same time RITTER (*Berl. klin. Wochenschr.*, 1892, p. 1276; 1896, No. 47, p. 1040, and No. 48, p. 1069) described a diplococcus which did not correspond to that of Cohn and Neumann. The work of the earlier investi-

gators, LETZERICH (*Virch. Arch.*, 1874, Bd. ix.), TSCHAMER (*Jahrb. f. Kinderheilk.*, 1876, Bd. x.), BURGER (*Berlin. klin. Woch.*, 1883), MONSORRO (*Rio de Janeiro*, 1883), is unimportant. KOPLIK (*Centralb. für Bakteriol. u. Parasitk.*, vol. xxi. p. 222) reports the examination of sixteen cases occurring in his service at the Good Samaritan Dispensary, New York. In thirteen of these cases he found, both in cover-glass preparations of the sputum and in culture, a bacillus which, when stained by Löffler's alkaline methylene-blue, appeared as a short, slender rod, thinner than the diphtheria bacillus and one-third its length. It had a beaded appearance, and in old culture showed distinct clubbed ends, which stained a dark blue. The organism was motile and did not form spores In sputum the bacillus was in immense numbers in the small whitish masses which occur, and was often enclosed in the pus-cells. To make cultures the sputum was collected in sterile Petri dishes, and after standing one hour the small clumps were picked out and placed on culture media. Coagulated hydrocele fluid was found to be the best culture medium. In pure culture the organism grew in a fine-pointed layer of pearl-white colonies. If one-third its bulk of glucose bouillon was added the colonies appeared cream-colored, resembling somewhat those of the diphtheria bacillus. It could be grown anaërobically, and did not liquefy gelatin. White mice inoculated with 0.5 c.cm. of fresh bouillon culture died at the end of a week, and the organism could be recovered from the blood. In some cases the spleen was enlarged. An inoculation of 2 c.cm. killed in twenty-four hours with general œdematous infiltration of subcutaneous tissues of back. Subcutaneous injections in rabbits and guinea-pigs were negative. Intravenous injections of old culture in rabbits produced pyæmia or suppuration of the joints. There were no lesions of the lungs or characteristic convulsive symptoms in any of the animals. In uncomplicated cases the bacillus was found in immense numbers, sometimes in pure culture. In cases with bronchitis or broncho-pneumonia, it was generally overgrown by the pneumococcus or the streptococcus. The writer considers that it corresponds to the bacillus described by Afanassieff; and that while more observations are needed to determine definitely its relations to whooping-cough, it must, nevertheless, be considered as an important organism in the bacteriological consideration of this disease.

CZAPLEWSKI and HENSEL (*Centralbl. für Bakteriol. und Parasitk.*, December 22, 1897) report the examination of the sputum in forty-four cases. Their methods consisted in staining cover-slip preparations with carbol glycerin fuchsin, and in making cultures from sputum lumps after washing in peptone solution to get rid of other organisms which might be present. They describe a short bacillus with distinctly staining, rounded ends, sometimes occurring in short chains, commonly in pairs, and often so small as to resemble cocci. This organism occurred constantly in the sputum, generally in immense numbers, and increased in numbers as the disease advanced. It occurred both free and in pus-cells. They obtained it frequently in pure culture, though in complicated cases it was difficult to avoid the streptococcus and the pneumococcus. It grew well on all the ordinary media except potato. On agar the growth appeared as small, dew-like, pointed colonies. The bacillus had the same general characteristics as the bacillus described by Koplak, with the exception of motility. They believe that Koplik's bacillus

is identical with theirs; and that Burger also described it in 1883. Their animal inoculations were negative. The bacillus was demonstrated in thirty cases. [Both from their description and from the excellent micro-photographs accompanying the article we are strongly reminded of a small diplobacillus commonly met with in routine examination of the throat in diphtheria.]

C. SPENGLER (*Deutsche med. Wochenschr.*, December 23, 1897), in some remarks on the communication of Czaplewski and Hensel, states that he found a similar bacillus to be commonly present in the sputum in an epidemic of whooping-cough which he had studied three years previously. He had delayed reporting his observations until another epidemic should furnish him an opportunity to confirm his results. He questions the methods of the above-named authors, and points out contradictions in the descriptions. The bacillus found by him agrees in general with the description which they give.

Typhoid Infection without Intestinal Lesions.—SIMON FLEXNER and NORMAN MCL. HARRIS (*Johns Hopkins Bulletin*, December, 1897) report a case of typhoid septicæmia in a patient who died of gangrene of the lung with pyo-pneumothorax. There was nothing in the clinical history of the case to suggest typhoid. The patient was admitted to the hospital two days before death. There was evident pneumothorax on the right side, and the blood-count gave 18,000 leucocytes. The only condition shown by the autopsy was thrombosis of a branch of the pulmonary artery, gangrene of the lung, and perforation of the pleura. There was no glandular enlargement anywhere, and although the spleen is described as acute splenic tumor, it weighed but 160 grammes, which is well within the variations of normal. Cultures from the blood and various organs gave bacilli which responded to all the tests for the bacillus typhosus, including the serum reaction. On histological examination of the organs none of the tissue changes common to typhoid were present.

Notes on an Experimental Investigation Into the Growth of Bacillus Typhosus in Soil.—JOHN ROBERTSON (*British Medical Journal*, January 8, 1898), after searching with negative results for the typhoid bacillus in the soil of a "typhoid area" (consisting of a group of seven cottages where eighteen cases of typhoid fever had occurred during eight and a half years), attempted to ascertain if it were possible for the organism to grow in the soil, and if so, under what circumstances. On May 10th he removed the vegetation from three patches of earth, each eighteen inches square, and inoculated each patch with 200 c.c. of a twenty-four-hour bouillon culture of the typhoid bacillus; one on the surface, one at a depth of nine inches, and the third at a depth of eighteen inches. No vegetation was allowed to grow on the patches. The soil was a clayey loam with strata of sand and clay below. The ground water level was high, often reaching to within three feet of the surface. On August 26th, after a season of comparatively dry weather (total rainfall 5.2 inches), and an abundance of sunshine, cultures were taken from each patch; and in each case the organism was found in sufficient numbers to indicate that new growth had taken place. On October 20th, after much wet weather, cultures were again taken with a similar result.

On November 27th, after cold, damp weather, the temperature often being below 32° F. (for one week averaging 23° F.), cultures were taken for a third time, with negative results. A second group of three similar patches planted in August also gave negative results on November 27th. From January until June, at intervals of two weeks, the second group of patches received dilute organic solutions (beef-tea, etc.), which the first group did not. On June 3d and July 11th each of these patches so fed showed the presence of the bacillus typhosus, while the others did not. That no lateral extension of the growth took place was shown by negative results in culture taken from soil at distances of one, three, and nine feet from the patches. As to the effect of sunlight, it was shown that only organisms directly exposed were destroyed. Cultures at a depth of one-sixteenth of an inch were positive. The methods used for identifying the typhoid bacillus were those commonly used, including the serum reaction. The writer concludes that the typhoid bacillus is capable of growing rapidly in certain soils, and under suitable conditions may survive from one summer to another. He compares the patches which he fed artificially with organic solutions to soil which receives a large amount of organic material through defective drainage; and considers the nutrient material or natural culture medium thus afforded as the essential element in the continuance of the organism in the soil.

A New Form of Serum Reaction with Colon Bacillus and Proteus Bacillus.—Previous work has shown (PFANDLER, *Centralblatt für Bacteriologie*, January 19 and 28, 1898) that an agglutinating reaction may occur in colon infections, but that it is rare and poorly marked. The author investigated the serum reaction in eight cases of colon infection, of which six were cystitis where the colon bacillus was isolated from the urine, the seventh was an enteritis with a general colon infection, and the eighth was a general peritonitis from intestinal perforation, and the organism was obtained at the time of operation. A case of proteus infection giving the proteus bacillus in the urine and a case of enteritis giving the bacillus lactis aerogenes in the urine and stools complete the series. Rather large amounts of blood were drawn and the serum obtained by centrifugalization. Three loops of a twenty-four-hour culture of the organism upon agar were mixed in a tube of bouillon, and this suspension was mixed with the serum in proportions of 1 to 10, 1 to 30, 1 to 50, and 1 to 100. Hanging-drop preparations were made of each dilution upon a hollow slide. Serum-free controls were made for comparison. No reaction occurred after two hours, but on standing for twenty-four hours it was found that, while the control drops had not altered their appearance, except by multiplication of the bacilli, the serum mixture showed the bacilli as very fine threads which were coiled around each other, forming distinct balls. In the weaker dilutions the clumps were isolated from each other or connected by fine threads. Between these knots the fluid was quite free from organisms, and the cells and knots themselves showed no trace of motility. Individual bacilli or very small groups of these were rarely found and were surrounded by the labyrinth of threads. In the macroscopic preparations of serum and bouillon there occurred a gross reaction in which the uniform clouding was replaced by partial clearing of the medium. Inoculations of these clumps upon agar gave back the colon

bacillus without change in its morphology. This reaction occurred only when the serum and the bacillus came from the same individual. A second essential for the reaction is that the patient should have a fever temperature. This is explained by the author by the asumption that a substance is formed in the body which will act upon the bacilli in the characteristic way only if the infection is intense, and the most common index of intensity is fever.

Immunity and Serotherapy against Yellow Fever.—SANARELLI (*Annales de l'Institut Pasteur*, October, 1897) says that most of the autopsies which he made in yellow fever were made shortly after death, and he often found the blood in the heart not coagulated, and collected the serum. The appearance of the serum varies: it may be transparent and clear, or slightly colored by blood or bile admixture. The yellowish serum may take a greenish tint by exposure to light. The serum of the cadaver produces the phenomena of agglutination on cultures of the bacillus icteroides, but the reaction is very variable. It has no inhibiting action against the organism. He obtained a considerable amount of serum from a convalescent eleven days after recovery, the man being still jaundiced. The serum was greenish, limpid, and transparent. It produced agglutination very slowly, and had a feeble inhibiting action against the organism when inoculated. Simultaneous injection of the serum with the virus was effective, but there was no effect when the serum was injected twenty-four hours before the inoculation. The organism is not destroyed in the serum, but remains alive for a long time. Other sera had no action. Antidiphtheritic serum and antityphic serum had no agglutinating action on the bacillus. Immunity against the disease is conferred in man by an attack. In experiments on the production of artificial immunity he gave up the use of rabbits, sheep, and goats on account of their sensitiveness to the organism. The immunity experiments were carried out on the guinea-pig, the dog, and the horse. The guinea-pig is difficult to immunize. He used for some weeks small doses of the filtered cultures or the pleural and peritoneal exudation of a goat killed by inoculation with the bacillus. After about one month of the treatment, and some days after the last injection, 0.1 c.c. of the virus was withstood. This quantity is absolutely fatal to the non-treated animal. During the treatments the weight of the animal reduces and by following the oscillation of this the treatment may be increased or diminished. Even with the greatest care these vaccinations produce a high mortality. In most cases it takes six or seven months before an animal is thoroughly immunized. The dog can be immunized much more rapidly than the guinea-pig. Immunity can be produced simply by injecting filtered cultures or cultures sterilized by ether. This proceeding produces infiltration and large purulent collections in the tissue. After this come the intravenous injections. The dogs are extremely sensitive to these injections; they vomit and have fever for several days. In seven or eight months they have an immunity against toxic doses of the bacillus. No matter how well vaccinated, the dogs are not proof against very large doses of the toxins. The beef stands subcutaneous injections better than the horse, but cannot stand the intravenous injections. The horse is given small doses (5 to 10 c.c.) of the filtered culture subcutaneously. These injections are followed by elevation of temperature,

which lasts several days. During this period the injections are discontinued. Intravenous injections should begin with small doses of the filtered cultures. They are well tolerated, and only produce a slight elevation of temperature. After each injection there are trembling and dyspnœa. Care must be taken not to inject outside of the vein. Five or six months after the beginning of the treatment injections with living cultures may be attempted. The production of immunity is in any case a slow and painstaking matter. He kept up the vaccination so long that some of the animals could be considered as hyper-vaccinated. One c.c. of the serum of a guinea-pig so inoculated was effective when injected twenty-four hours before a fatal dose of the virus. Of twenty-six guinea-pigs inoculated with the virus, twenty were treated with serum; six not. The six died in six to twelve days; of the twenty treated, three died between the tenth and sixteenth day. The dog-serum was strong. It gave the phenomena of agglutination to a marked degree. The efficacy of the serum of the dog does not seem due to antitoxic properties. The preventive action is more efficacious than the curative action. Ill success was met with in the production of horse-serum—two of the horses dying. A third horse gave a serum which was feeble.

[The sera were only tried with the guinea-pig—an animal which is rather resistant to the virus—and the results obtained, though interesting, do not give much hope for the application of serum-therapy against the disease.]

Staining of Müller's Granules.—NICHOLLS (*Philadelphia Medical Journal*, February 26, 1898) reports the successful staining of the "dust bodies" of the blood, first described by Müller in 1896. His method consists in adding minute quantities of Ehrlich's neutrophile stain to 0.5 per cent. saline solution and allowing it to flow under a cover-slip of fresh blood sealed with vaseline. Eosine added in the same way stains the large granules. This he considered as completing the chain of evidence necessary to prove the theory of Stokes and Wegefarth.

The Presence in the Blood of Free Granules Derived from Leucocytes, and their Possible Relations to Immunity.—STOKES and ARTHUR WEGEFARTH (*Johns Hopkins Hospital Bulletin*, December, 1897) refer to the observations of Müller, who described small round colorless bodies as constantly present in the blood of both healthy and diseased persons. Müller did not attempt to explain the origin or composition of these bodies. The authors were able to demonstrate the presence of such bodies in blood taken from the lobe of the ear in 500 individuals, of whom 100 were healthy. They found the granules were most easily seen in artificial light obtained from a Welsbach burner. The granules are increased in number by the addition of dilute solutions of acetic acid to the blood, and they are believed to come from the granular leucocytes of the blood, either the oxyphile or the eosinophile. A number of observations have shown that the bactericidal powers of the blood-serum are diminished when it is collected without leucocytes, and that such serum may be made bactericidal by the addition of leucocytes. The authors filtered dog-serum through porcelain filters, and found a decided difference in the bactericidal powers of the serum before and after filtration. The unfiltered serum produced agglutination in the cholera, Finkler-Prior,

and typhoid bacilli in from fifteen to twenty minutes, and the filtered serum showed no action in three hours. The filtered serum had no action on the same organisms, while the unfiltered destroyed them. They were not able to perfectly restore the bactericidal action of the filtered serum by the addition of leucocytes containing the granules. They advance the theory that the bactericidal power of the leucocytes of the blood, and of the serum of man and many animals is due to the presence of specific granules, especially the eosinophilic and the neutrophilic. When called upon to resist the action of invading bacteria the granular leucocytes can give up their granules to the surrounding fluids or tissues.

Miliary Tuberculosis.—RIBBERT (*Deut. med. Woch.*, December 30, 1897) replies to Weigert's criticisms of Wild's conclusions with regard to the occurrence of miliary tuberculosis. Wild does not accept Weigert's doctrine that all the bacilli necessary for the production of miliary tuberculosis are derived from tubercles of veins or lymph vessels, regarding it as highly improbable that such great numbers as must be required for this could enter through a lesion so slight as in most cases to be invisible macroscopically. To Weigert's objection that in some cases but few or no tubercle bacilli may be found in the blood, Ribbert replies that he believes with Wyssokowitsch, that the bacilli may disappear from the blood, multiply in the organs to which they have been carried, and later again enter the circulation. Ribbert believes that the multiplication of the tubercle bacilli in the blood is dependent upon various conditions: a high degree of toxicity of the bacilli, diminution of the resistance of the invaded organisms, and predisposition. He holds that the connection between tuberculosis of veins or lymph vessels and miliary tuberculosis, in the sense that all the bacilli necessary for the general infection come from one point of entrance, is not proven, and that in most cases only a few enter the blood. He believes that proliferation of the bacilli takes place in the blood under certain conditions in those individuals who are predisposed, and in others whose resistance has become diminished.

Notice to Contributors.—All communications intended for insertion in the Original Department of this Journal are only received *with the distinct understanding that they are contributed exclusively to this Journal.*

Contributions from abroad written in a foreign language, if on examination they are found desirable for this Journal, will be translated at its expense.

Liberal compensation is made for articles used. A limited number of reprints in pamphlet form, if desired, will be furnished to authors in lieu of compensation, *provided the request for them be written on the manuscript.*

All communications should be addressed to

DR. EDWARD P. DAVIS, 250 South 21st Street, Philadelphia, U. S. A.

Or

DR. HECTOR MACKENZIE, 59 Welbeck St., Cavendish Sq., London, W., Eng.

CONTENTS.

ORIGINAL COMMUNICATIONS

PAGE

An Inquiry into the Etiology of Cancer, with some Reference to the Latest Investigations of the Italian Pathologists. By ROSWELL PARK, A.M., M.D. . . 503

A Contribution to Our Knowledge of the Etiology of Inflammations of the Accessory Sinuses of the Nose. By W. T. HOWARD, JR., M.D., and J. M. INGERSOLL, M.D. 520

A Simple Method for the Sterilization of Catgut. By CHARLES HARRINGTON, M.D. 544

Surgical Treatment of Epilepsy, with a Report of Fourteen Cases. By ANDREW J. MCCOSH, M.D. 549

REVIEWS.

Diseases of the Eye. By Edward Nettleship, F.R.C.S. With a Supplement on Color-blindness by William Thomson, M.D. 574

A Clinical Text-book of Surgical Diagnosis and Treatment. By J. W MacDonald, M.D. 575

The Practice of Surgery. By H. R. Wharton, M.D., and B. F. Curtis, M.D. . . 576

Text-book of Nervous Diseases. By Charles L. Dana, A.M., M.D. . . . 576

A Manual of Clinical Diagnosis by Means of Microscopic and Chemical Methods, By Charles E. Simon, M.D. 578

Orthopedic Surgery. By James E. Moore, M.D. 579

Clinical Methods: A Guide to the Practical Study of Medicine. By Robert Hutchison, M.D., M.R.C.P., and Harry Rainy, M.A., F.R.C.P. Edin., F.R.S.E. . 579

Lectures on the Malarial Fevers. By William Sydney Thayer, M.D. . . . 580

A System of Practical Medicine. Edited by A. L. Loomis, M.D., and W. G. Thompson, M.D. 581

PROGRESS OF MEDICAL SCIENCE.

THERAPEUTICS.

PAGE

Inhalations of Vinegar to Control Nausea and Vomiting after Chloroform 583

Note on Diastatic Preparations . . 583

Treatment of Chronic Enteritis . . 584

Therapeutic Value of Spleen Extract 584

Generalized Erythema following the Ingestion of Calomel . . . 586

Potassium Permanganate as an Antidote for Opium and Morphine . 686

PAGE

Some Effects of Cannabis Indica in Large Doses 587

Strychnine Poisoning . . . 587

Liquid Benzoin for Benzoinating Lard 587

Electrotherapy in Neurasthenia . 587

Antipyrin in Epidemic Influenza . 588

Results in Treatment of Tuberculosis with Anti-tuberculosis Serum . 588

Tuberculin R. 590

MEDICINE.

Myelin in Sputum 590

Diuresis, instead of Critical Sweat, in Pneumonia 591

Muscle-callus 591

Lipuria in Hysteria 592

Chlorosis 592

Hemicrania and Basedow's Disease . 593

Traumatic Myocarditis . . . 593

Tuberculous Endocarditis . . . 594

Dental Caries and Soil . . . 594

Typhoid Fever without Intestinal Lesions 594

Epilepsy and Diabetes Mellitus . 595

Spontaneous Rupture of a Fatty Heart 597

The Use of Sudan III. as a Staining in Clinical Microscopy . . . 597

SURGERY.

	PAGE		PAGE
Lumbar Nephropexy without Suturing	598	The Forcible Correction of Deformity of Pott's Disease	603
Glandular Enlargements of the Neck	599	Ossifying Osteoperiostitis of Metatarsals	604
Straightening of the Spine	600	Treatment of Tumors of Large Intestine	605
A Case of Ligature of the Innominate Artery for Aneurism	600	Treatment of Exophthalmic Goitre	605
Mortality of Acute Intussusception	601	Trephining of the Mastoid	606

OPHTHALMOLOGY.

Trachoma Treated with Iodine	607	Optic Atrophy following Sexual Excess	609
Membranous Keratitis	607	Treatment of Trachoma	609
Transient Hemianopsia	608	Strabismus	610
The Pupils in Multiple Sclerosis and Syphilitic Disease of Nerve Centres	608	Colored Vision after Exposure to Excessive Light and after Cataract Extraction	611
Entropion Operation	609		

OBSTETRICS.

The High Application of Forceps in Cases of Contracted Pelvis	612	Laceration of the Rectum above the Sphincter in Normal Labor	613
Influence of Morphine and Ether upon Labor Pains	612	Extrauterine Pregnancy and Abdominal Section	613
Cæsarean Section with Transverse Incision of the Fundus for Carcinoma	612	Secretion of Urine by the Fœtus	614
Cæsarean Section with Transverse Incision of the Fundus	613	Pregnancy and Labor in which Amputation of Cervix has been Performed	614
		Symphysiotomy	615

GYNECOLOGY.

Perforation of the Uterine Wall during Curettage	616	Influence of Menstruation on Chronic Psychoses	618
Palpation of the Uterine Cavity	617	Metrorrhagia due to Liver Disease	618
Ascites in Young Girls	617	Rupture of the Vagina during Coitus	618
Pregnancy after Transplantation of the Ovary	617	Malignant Fibro-cyst of the Uterus	619
Injuries due to Bicycling	617	Carcinomatous and Sarcomatous Degeneration of a Uterine Fibroma	619
Intra-pelvic Displacement of Appendix	618	Histology of the Prolapsed Uterus	619

PÆDIATRICS.

A Case of Pemphigus Neonatorum	620	The Pseudo-membranous Anginas of Scarlatina	621
Acute Nephritis Complicating Mumps	620		

PATHOLOGY AND BACTERIOLOGY.

Syringomyelia	622	Phagocytosis in Relapsing Fever	624

HYGIENE AND PUBLIC HEALTH.

Treatment of Town Sewage	625	Plague Regulations	627